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COMPARATIVE EFFECTS OF SULPHATHIAZOLE AND SULPHANILAMIDE ON SMALL-POX

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Introduction

In a previous article on "Sulphanilamide in Small-Pox" in this Journal, it was concluded that sulphanilamide attenuated the virulence of virus as evidenced by reduction of toxæmia, abortion of the eruptions, and lowered mortality rate particularly in the more virulent type, *viz.,* confluent variety of the disease. It also reduced incidence of complications caused by invasion of secondary organisms.

Various workers including the author of the article had mentioned about the beneficial results of the investigations with sulphanilamide in the treatment of small-pox. Some have tried preparations like sulphapyridine or occasionally sulphathiazole but none used them extensively to compare their relative merits conclusively. The present author who had given an extensive trial to sulphanilamide in 1942, has now tried sulphathiazole with a view to comparing its effects in small-pox with those observed with sulphanilamide.

Material

The total number of cases investigated was 202 out of which 101 were treated with sulphathiazole and an equal number with sulphanilamide. The cases that expired within 48 hours of admission were excluded for obvious reasons. The administration of the drugs was commenced in various stages of the disease for which the cases were divided into the following three groups:

Group 1—In which the drugs were started in the papular stage of the rash *i.e.,* on the third or fourth day of the illness.

Group 2—In which treatment was commenced in the vesicular stage of the rash *i.e.,* on the fifth or sixth day of the illness.

Group 3—In which treatment was commenced in the pustular stage of the rash *i.e.,* after eighth day of the illness. The dosage of the drugs in each case was one tablet (0·5 gm.) four hourly in adults. It was varied according to the age of the patient. The tablets were discontinued when the stage of scab formation commenced. The small dose of the drug was given for prophylactic purpose and not to effect cure of the disease.

Toxæmic manifestations like insomnia, delirium, stupor, vomiting, dehydration and failure of peripheral circulation were watched in each group, the incidence of septic complications like abscesses, ulcers, bedsores and respiratory complications like bronchitis and pneumonias were recorded as met with in patients of each of the groups.

Results

(1) *Mortality rate*—(*a*) The total mortality amongst the 202 cases was 21·2 per cent. It was 18·8 per cent in sulphathiazole series whereas in sulphanilamide series it was 23·7 per cent.

(*b*) In the more fatal form of the disease *viz.,* its confluent variety, the mortality rate in Group 1 *i.e.,* when the drug was commenced in the papular stage, was 40·0 per cent and 53·3 per cent in sulphathiazole and sulphanilamide series respectively (*vide* Table I).

In Group 2 *i.e.,* when the drug was commenced in the vesicular stage it was 53·8 per cent and 63·5 per cent in sulphathiazole and sulphanilamide series respectively (*vide* Table II).

In Group 3 *i.e.,* when the drug was commenced in the pustular stage there was only one case treated on each side and none died (*vide* Table III).

(*c*) In less severe form *i.e.,* semi-confluent variety of the disease, the mortality rate in Group 1 (papular stage) was 26·5 per cent and 31·5 per cent in sulphathiazole and sulphanilamide series respectively (*vide* Table I).

In Group 2 (vesicular stage) it was 6·6 per cent and 13·3 per cent in sulphathiazole and sulphanilamide series respectively (*vide* Table II).

In Group 3 (pustular stage) it was nil on both sides (*vide* Table III).

(*d*) In the most mild and discrete form of the disease the mortality rate in all the three groups was nil in both the series.

(*e*) The total mortality in all the three groups together in sulphathiazole and sulphanilamide series with confluent variety was 44·9 per cent and 52·2 per cent respectively, that in semi-confluent variety was 13·3 per cent and 17·3 per cent in the two series respectively and there was no mortality in all cases of the groups with discrete variety of both the series (*vide* Table IV).

Thus, the mortality rate is definitely lowered under sulphathiazole treatment as compared with that

under sulphanilamide treatment and the observation is appreciably noted in the more severe form of the disease *viz.*, confluent variety.

(2) *Duration of the rash*—The duration of the rash during its evolution from papular stage to the scab formation as well as till all the scabs were separated was very much reduced by the administration of sulphonamides. When the results of the two drugs of the sulphonamide group were compared for their relative merits it was found that sulphathiazole was more potent in this respect than sulphanilamide. It was further observed that in the majority of sulphathiazole treated cases the scab had fallen off without leaving behind superficial ulcers, whereas in sulphanilamide treated cases, the separation of scabs generally revealed superficial ulcers which took about two days to heal completely.

(3) *Abortion of the rash*—The rash aborted in 53·4 per cent and 35·6 per cent under sulphathiazole and sulphanilamide therapy respectively (*vide* Table IV). In such cases most of the vesicles were found to dry up without passing through the pustular stage. Even the duration of the individual stage of the rash was found to be shorter. The percentage of abortion of the rash was found to be increased most markedly when the drugs were commenced in the early papular stage *viz.*, the 71·4 per cent and 45·2 per cent of cases of the two series respectively (*vide* Table V). In these cases the vesicles were found to be smaller, thinner and more superficial while the pustules looked less turbid, especially in sulphathiazole series than in sulphanilamide ones.

(4) *Toxæmia*—Toxæmic manifestations like insomnia, delirium, stupor, vomiting, dehydration and peripheral circulatory failure were observed to be less common in sulphathiazole than in sulphanilamide series *viz.*, 28·7 per cent and 33·6 per cent respectively (*vide* Table IV). Toxæmia was found to be very much reduced when the drugs were administered in the early papular phase of the disease and it was found to be particularly so with sulphathiazole series. The drug-toxæmia was not noticed in any of the cases. This might be due to the small dosage in which the drugs were administered or might be due to the administration of other supporting drugs such as nicotinic acid tablets, sodium bicarbonate and liver therapy prescribed along with the chemotherapeutic agents.

(5) *Complications*—(*a*) Septic complications like abscesses and ulcers were much less in sulphathiazole treated cases than in sulphanilamide treated cases *viz.*, 10·9 per cent and 17·8 per cent respectively (*vide* Table IV). Corneal ulcers were noticed in three cases treated with the latter drug only and arthritis and bedsores were not observed in any of the cases. These respective figures were found to be further reduced when the treatment was commenced in the early papular stage.

(*b*) The lung complications were found to be 4·9 per cent and 11·8 per cent only with sulphathiazole and sulphanilamide respectively, showing conclusively the more potent prophylactic action of the former drug over the latter, much more so when the drug was given in the early papular stage of the disease (*vide* Table IV).

(*c*) The incidence of secondary fever was found in 20·5 per cent and 32·8 per cent of the cases treated with sulphathiazole and sulphanilamide respectively. There was not a single case of drug fever observed.

(*d*) Seven cases of hæmorrhagic type of small-pox were treated, out of which 3 were treated with sulphathiazole and four with sulphanilamide. Out of the former three, 2 cases were of confluent type and 1 semi-confluent type. Both confluent cases expired and the semi-confluent one cured. Out of the latter four cases, 3 were of confluent type and 1 semi-confluent type. Out of them 2 confluent cases expired and the rest cured. All these cases were very toxic with marked dehydration from the beginning with extensive hæmorrhages underneath the skin. A definite opinion about the comparative effects of the chemotherapeutic drugs cannot be given as the cases were very few but the noteworthy fact was that with sulphanilamide there was less extensive hæmorrhage in the rash than with sulphathiazole.

Summary and Conclusions

(1) The results of therapeutic actions of sulphathiazole and sulphanilamide on small-pox in 202 patients equal number of which was treated with these drugs separately are reviewed in order to find out which of the drugs is comparatively more potent in modifying the evolution and course of the different stages of the disease, reducing the incidence of complications and mortality rate, etc.

(2) Sulphathiazole was found to be more potent in reducing the virulence of the virus as evidenced by abortion of the rash, shortening of the period of scab formation and reduction of the toxæmia. The patients were found to be fit for discharge *i.e.*, free of all the scabs over the body much earlier with the treatment by sulphathiazole than with sulphanilamide. The most important fact to be noted was that the scabs in the case of former therapy were more papery, superficial and after their separation left behind superficial scars without pitting or disfiguration, while with sulphani lamide the scabs after detachment left behind superficial ulcers which took about 2 days to heal completely.

(3) Reduction of toxæmic manifestations accompanying the acute course of the disease was much more evident by sulphathiazole than by sulphanilamide. Even the duration and degree of toxæmia was found to be shorter and milder and their incidence was less with sulphathiazole than with sulphanilamide.

(4) The septic complications were found to have lower incidence under sulphathiazole than under sulphanilamide therapy. Corneal ulcers were observed in cases treated with the latter drug only. Secondary pyrexia was observed in a lesser number of cases and its duration was shorter with sulphathiazole than with sulphanilamide. The lung complications were also much rarely noticed under sulphathiazole than under sulphanilamide treatment.

(5) The average course of treatment extended over the period of 4·8 and 7·1 days in case of sulphathiazole and sulphanilamide respectively. So the total

TABLE I—SHOWING THE EFFECTS OF SULPHATHIAZOLE AND SULPHANILAMIDE ADMINISTERED IN THE PAPULAR STAGE IN THE GROUP I SERIES

	SULPHATHIAZOLE				SULPHANILAMIDE			
	Confl.	*Semi-Confl.*	*Disc.*	*Total*	*Confl.*	*Semi-Confl.*	*Disc..*	*Total*
No. of cases treated	15	19	8	42	15	19	8	42
Rash aborted	10(66·6%)	14(73·5%)	6(75·0%)	30(71·4%)	5(33·3%)	10(52·5%)	4(50·0%)	19(45·2%)
Maximum toxæmia	6(40·0%)	5(26·3%)	Nil.	11(26·2%)	5(33·3%)	6(31·5%)	1(12·5%)	12(28·6%)
Septic complications	2(13·3%)	Nil.	Nil.	2(4·8%)	3(20·0%)	2(10·5%)	Nil.	5(12·0%)
Lung complications	1(6·6%)	1(5·2%)	Nil.	2(4·8%)	3(20·0%)	2(10·5%)	Nil.	5(12·0%)
Corneal ulcers	Nil.	Nil.	Nil.	Nil.	2(13·3%)	Nil.	Nil.	2(4·8%)
Average duration in days of pustular stage in unaborted cases	3·0	2·8	1·3	2·3	3·6	3·0	2·5	3
Mortality	6(40·0%)	5(26·5%)	Nil.	11(26·2%)	8(53·3%)	6(31·5%)	Nil.	14(33·3%)

TABLE II—SHOWING THE EFFECTS OF SULPHATHIAZOLE AND SULPHANILAMIDE ADMINISTERED IN THE VESICULAR STAGE IN THE GROUP II SERIES

	SULPHATHIAZOLE				SULPHANILAMIDE			
	Confl.	*Semi-Confl.*	*Disc.*	*Total*	*Confl.*	*Semi-Confl.*	*Disc..*	*Total*
No. of cases treated	13	15	10	38	13	15	10	38
Rash aborted	8(61·1%)	10(66·6%)	6(60·0%)	24(63·1%)	6(46·2%)	6(40·0%)	5(50·0%)	17(45·0%)
Maximum toxæmia	7(53·8%)	4(26·9%)	1(10·0%)	12(31·6%)	6(46·2%)	6(40·0%)	2(20·0%)	14(36·8%)
Septic complications	2(15·4%)	3(20·0%)	Nil.	5(13·2%)	4(30·7%)	3(20·0%)	1(10·0%)	8(21·0%)
Lung complications	2(15·4%)	Nil.	Nil.	2(5·3%)	4(30·7%)	2(13·3%)	Nil.	6(15·9%3
Corneal ulcers	Nil.	Nil.	Nil.	Nil.	1(7·7%)	Nil.	Nil.	1(2·6%)
Average duration in days of pustular stage in unaborted cases	3·5	3·3	2·0	2·9	4	3·5	2·5	3·3
Mortality	7(53·8%)	1(6·6%)	Nil.	8(21·0%)	8(61·5%)	2(13·3%)	Nil.	10(26·3%)

TABLE III—SHOWING THE EFFECTS OF SULPHATHIAZOLE AND SULPHANILAMIDE ADMINISTERED IN THE PUSTULAR STAGE IN THE GROUP III SERIES

	SULPHATHIAZOLE				SULPHANILAMIDE			
	Confl.	*Semi-Confl.*	*Disc..*	*Total*	*Confl.*	*Semi-Confl.*	*Disc.*	*Total*
No. of cases treated	1	11	9	21	1	11	9	21
Rash aborted	Nil.	Nil.	Nil.	Nil.	Nil.	Nil.	Nil.	Nil.
Maximum toxæmia	1(100·0%)	4(36·4%)	1(11·1%)	6(28·6%)	1(100·0%)	6(54·5%)	1(11·1%)	8(38·1%)
Septic complications	1(100·0%)	3(27·3%)	Nil.	4(19·0%)	1(100·0%)	4(36·4%)	Nil.	5(23·8%)
Lung complications	Nil.	1(9·1%)	Nil.	1(4·8%)	Nil.	Nil.	Nil.	1(4·8%)
Corneal ulcers	Nil.	Nil.	Nil.	Nil.	Nil.	1(9·1%)	Nil.	Nil.
Average duration in days of pustular stage in unaborted cases	4·5	3·5	3·0	3·6	4·5	4·0	3·5	3·9
Mortality	Nil.	Nil.	Nil.	Nil.	Nil.	Nil.	Nil.	Nil.

TABLE IV—SHOWING THE EFFECTS OF SULPHATHIAZOLE AND SULPHANILAMIDE ADMINISTERED IN ALL COMBINED STAGES OF THE DISEASE.

	SULPHATHIAZOLE				SULPHANILAMIDE			
	Confl.	*Semi-Confl.*	*Disc.*	*Total*	*Confl.*	*Semi-Confl.*	*Disc..*	*Total*
No. of cases treated	29	45	27	101	29	45	27	101
Rash aborted	18(62·1%)	24(53·3%)	12(44·4%)	54(53·4%)	11(37·9%)	16(35·5%)	9(33·3%)	36(35·6%)
Maximum toxæmia	14(48·3%)	13(28·8%)	2(3·7%)	29(28·7%)	12(41·4%)	18(40·0%)	4(14·8%)	34(33·6%)
Septic complications	5(17·2%)	6(13·3%)	Nil.	11(10·9%)	8(27·6%)	9(15·5%)	1(3·7%)	18(17·8%)
Lung complications	3(10·4%)	2(4·4%)	Nil.	5(4·9%)	7(24·1%)	5(11·1%)	Nil.	12(11·8%)
Corneal ulcers	Nil.	Nil.	Nil.	Nil.	3(10·3%)	Nil.	Nil.	3(2·9%)
Average duration in days of pustular stage in unaborted cases	3·6	3·5	1·2	3·0	4	3·5	2·8	3·4
Mortality	13(44·9%)	6(13·3%)	Nil.	19(18·8%)	16(55·2%)	8(17·7%)	Nil.	24(23·7%)

dosage required was much less in case of the former drug than in the latter. The dosage followed in these series of cases was one tablet (0·5 gm.) four hourly and the respective drug was discontinued as soon as the scab formation commenced. The results obtained with the smaller dosage were more beneficial than those obtained with larger dosage mentioned in our previous article on the subject *viz.,* two tablets every four hours for the first 48 hours, two tablets every six hours for the next 48 hours and one tablet every four hours for the next 48 hours. Even taking into consideration the higher virulence and mortality rate of small-pox cases in the last epidemic of the disease when previous work was carried out it is to be noted that the percentage of mortality, septic complications and toxæmia were reduced to an appreciable degree.

(6) The mortality rate was found to be more reduced with sulphathiazole than with sulphanilamide and this fact was particularly striking in cases where the drugs were commenced in the early papular stage of the most severe form of the disease *viz.,* the confluent variety.

(7) The observations made in the previous investigations regarding the beneficial effects of previous vaccination were also confirmed during the study of the present series of cases. With sulphathiazole the mortality rate of the total number of unvaccinated cases was 24·6 per cent which was reduced to 13·6 per cent in previously vaccinated cases. With sulphanilamide the mortality rate of the total number of unvaccinated cases was 32·5 per cent which was reduced to 19·0 per cent in previously vaccinated cases. The septic complications were reduced from 12·5 per cent in the unvaccinated cases to 9·3 per cent in previously vaccinated ones under sulphathiazole and from 19·3 per cent in the unvaccinated cases to 16·5 per cent in the previously vaccinated ones under sulphanilamide. The lung complications were also reduced from 6·2 per cent in the unvaccinated to 3·9 per cent in the previously vaccinated cases with sulphathiazole and from 13·7 per cent in the unvaccinated to 8·3 per cent in the previously vaccinated cases with sulphanilamide. These figures conclusively prove the modifying influence of previous vaccination which, by inducing partial immunity helps in the further reduction of the mortality rate, septic complications and lung complications under chemotherapy.

(8) On comparing the mortality rate observed in the previous series of cases with that of the present ones, we find marked variation. The mortality rate in confluent variety noted in the previous work was 73·6 per cent with sulphanilamide. In the present series it was 48·3 per cent and 58·8 per cent in confluent variety of cases under sulphathiazole and sulphanilamide therapy respectively. In semi-confluent variety it was 20·7 per cent in the previous cases of the heavy epidemic, while it was now noted to be 13·3 per cent and 17·7 per cent with sulphathiazole and sulphanilamide therapy respectively. Thus, there is definite reduction in the total mortality rates in the present series of cases, a fact which can be explained by noting that the previous investigations were carried out during the heavy epidemic of virulent small-pox in the year 1942, whereas the present work was undertaken during the endemic exacerbation of small-pox in the year 1945 when there is comparatively higher immunity of the population and lower virulence of the virus.

References

Chakravarty and Ghosh—*Indian M. Gaz.,* **76**:220, 1941.
Chari, N. N.—*Ibid.,* **77**:91, 1942.
King C. and Rozario, K.—*J. Roy, Army M. Corps,* **71**:404, 1938.
MacCammon, W. C.—*J.A.M.A.,* **112**:1936, 1939.
Mallaya, B. G. and Chatterjee, B. C.—Report of the School of Tropical Medicine, Calcutta, 1940.
Moore, D. F.—*Trans. Roy, Soc. Trop. Med. & Hyg.,* **33,** 1940.
Patel, T. B. and Naidu B.—*Indian M. Gaz.,* **75**: 1940.
Pierna, S.—*J.A.M.A.,* **114**:931, 1940.
Wilkinson, P. B.—*Lancet,* **2**:67, 1942.
Vengsarkar, S. G. Poonen, T. N. and Walvalkar, S. A.—*J. Indian M. A.,* **11**:36, 1942.
Vengsarkar, S. G. and Rangnekar, J. V.—*J. Indian M. A.,* **13**:213, 1944.

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References

Amberson Jr.—Practitioners' Library. 1936. Vol. III.
Boyd—Pathology of Internal Diseases. 1944.
Brown and Sampson—Intestinal Tuberculosis. 1930.
Conybeare—Text-Book of Medicine. 1936. Published by E. S. Livingstone, Edinburgh.
Gloyne, S. Roodhouse—British Enclycopedia of Medical Practice. 1938. Published by Butterworth & Co. (Publishers) Ltd., London.
Goldberg—Clinical Tuberculosis, 1935.
Hurst—Prices' Text-Book of Medicine, 1944. Oxford University Press, London.
Maxwell—*Tubercle,* **17**:22, 1936.
Osler and McCrea—Principles and Practice of Medicine, 1936, D. Appleton & Co., London.
Shrikhande—*Indian M. Gaz.,* **71**:83, 1936.
Tribedi and Gupta—*J. Indian M. A.,* **11**:41, 1.
Ukil—*Indian M. Gaz.,* **77**:613, 1942.

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Some times there occur pallor, extreme nervousness and rapid pulse but these are only transitory in character.

Sacral anæsthesia when properly done is a safe and reliable method with practically no risk. It is not attended with any unpleasant postoperative complication as fall of blood pressure, headache, nausea, vomiting and distension of the bladder.

I have used the method so far from 1924 in not less than 400 cases and have done all types of operations mentioned above on the perinum with no risk whatsoever. It is the ideal method in perineal surgery of both the male and female and hence commends itself for much more liberal use in the hands of surgeons than at present.

MALNUTRITION AND ITS EFFECTS*

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The incidence of anæmia and malnutrition with its diverse effects has been rather high amongst the Indian patients evaçuated from the Eastern Army.

Out of a total of 6070 medical cases admitted to a Base General Hospital during the period of from February, 1944 to December 1944, 1257 cases had manifestations of nutritional deficiencies.

The chief clinical manifestations were diarrhœa, flatulent distension of the abdomen particularly after meals, glossitis, anæmia and marked emaciation. Some of the cases were indistinguishable from the syndrome of sprue or para-sprue described in text-books.

The object of this paper is to give a brief outline of these cases with a view to stimulate discussion on its ætiology and relationship with other deficiency syndromes.

Obviously, the state of nutrition of an individual will depend on adequate ingestion, absorption and utilisation of his food; and in order to assess the true value of nutrition it is essential to know whether enough and adequate food is being taken.

India is mainly an agricultural country and the average Indian is employed in manual occupation. Further, this country presents a special problem on account of highly industrialised state in certain places, diversity of population, physical habits, climatic variations and poverty.

Following the standard of requirements drawn up by the expert commission of League of Nations we can say that these people probably require 2800—3000 calories per diem; and this should be made up of:—

Proteins	..	..	65 gms.
Fats	..	..	45 to 60 gms. (1½-2 ozs. daily)
Vitamin	A	..	5000 International Units.
"	B	..	594 " "
"	C	..	2500 " "

An attempt was, therefore, made to investigate in detail the diet of these patients as elicited from case histories and here are some of the examples on the charts.

If we compare this with the type and nature of diet received by an Indian soldier in his various phases of life, namely at home, in peace stations, during field service and on patrols we can easily observe that the majority of these people have been brought up on an ill-balanced diet at home, that the diet was just on the threshold of adequacy (both in quality and quantity) if the consumer happens to be atta and meat eater and that it was low in proteins (of higher biological assay) in the case of vegetarians, more so if they are rice-eaters.

These factors corroborate two things on the clinical side:—

(*a*) High incidence of these states in South Indian vegetarians who are mostly rice-eaters (73 per cent). (*b*) In Middle East where malnutritional states were not so marked as in Eastern Army the incidence of anæmia was almost exclusively limited to Hindu vegetarians.

There is yet another difficulty added to these and that applies even to British soldiers subsisting on 'K' rations. It has been worked out that even if the diet contains 3000 calories daily, men are actually taking about 2300 calories, less than the generally accepted allowance for a man doing sedentary work. This is due to anorexia in the field which is constantly borne out from the case histories. Following possible explanations are suggested: an anxiety state resulting from the threat of enemy bombing, the effects of excessive heat and humidity and the dislike for some of the processed food of their rations of which they had become literally sick.

So we can say that there has been an (*i*) an inadequate intake during the period at home and this is shown by the poor nutritional conditions of many Indian recruits on enlistment, (*ii*) a difficulty of supply particularly of fruit and fresh vegetables in jungle warfare due to long line of communications. The feeding of patrol literally speaking has rightly been described as the "Quartermaster's nightmares", (*iii*) service is in areas hyperendemic to the incidence of malaria, dysentery and worm infestations (round worms and hookworms). Majority of these people get infected. The exceptional need for food of higher caloric value following such illnesses cannot be met with in forward units, (*iv*) further these intestinal affections lead to deficiencies of essential factors by:—

(*a*) Failure of absorption due to intestinal hurry or diminished absorbing surface.

(*b*) Failure of the bacteria to grow and synthesise vitamins. This failure being caused either by repeated attacks of diarrhœa, unsuitable alkaline media, or by ingestion of sulphonamide group of drugs (often given irregularly)—which reduces the natural flora of bowel, thus inhibiting the synthesis of many factors essential to the host *e.g.*, thiamin, members of B_2 complex—(pantothenic acid, folic acid, biotin, nicotinic acid and riboflavin etc.)

(*c*) There is another more obscure and complicated relationship between the gut and vitamins. Sydenstricker and his colleagues and also Petri and his associates have postulated an intrinsic factor produced by the stomach which acts in preventing pellagra; and he believes that the stomach is necessary for the synthesis of co-enzymes I and II from niacin, of flavo proteins enzymes from riboflavin, and of cocarboxylase from vitamin B_1; the latter is in agreement with the suggestion of Laurent and Sinclair that diminished phosphorylation of vitamin B_1 with failure of co-carboxylase formation may account for gastrogenous polyneuritis. Verzar believed that the failure to absorb fatty acids and glucose in the sprue-syndrome was due to failure of phosphorylation in the intestinal mucosa.

Since vitamin deficiencies produce disorders of the gut and disorders of the gut produce vitamin deficiencies, it is often difficult to decide which is the cart and which the horse.

(*v*) Effects of Low Acidity in the stomach—Test meal records are available for 150 cases. Of these 82 showed hypochlorhydria, 11 showed hyperchlorhydria, 19 showed achlorhydria (not histamin fast) and 38 showed normal curve.

* A lecture delivered before the Hyderabad-Dn. Branch of I.M.A. on 27-10 45.

In view of the high incidence of hypochlorhydria it is possible that this may have some effect, in its causation.

This hypochlorhydria may have been constitutional, result of previous intestinal affections or even the result of niacin deficiency itself.

The possible effects of hypochlorhydria are as follows:—

(*a*) Affects motility of the villi and decreases absorption of vitamin B content of the food. The stagnation and malabsorption will lead to abdominal discomfort and flatulance. (*b*) As a consequence of achlorhydria, migration and multiplication of intestinal flora. (*c*) Consequent breakdown of iron metabolism in relation to bone marrow and hæmoglobin—producing microcytic anæmia. (*d*) Hæmoglobin metabolism perverted to increasing amounts of porphyrin, bilirubin excretion reduced; bilirubin serum content increased. (*e*) Following liver damage bile acids decreased in turn reducing hydrotropy and fat absorption. (*f*) Unresolved fatty acids in the small intestine forming insoluble calcium salts; this affects the bones; and then the whole electrolyte equilibrium. (*g*) Increasing liver dysfunction; excessive porphyrinuria whereby Meissner's plexus is paralysed; motility of villi disturbed; breakdown of cytochrome enzymes and steatorrhœa.

In view of this it seems probable that there is a clear connection between the aetiology of this nutritional disorder, and that of sprue, idiopathic steathorrhœa and cœliac disease.

Clinical Manifestations

The signs and symptoms were so distinctive as to stamp themselves indelibly on the physician's mind. The association of glossitis, diarrhœa (with or without steatorrhœa), meteorism, and anæmia with emaciation was noted in various combinations. Five different groups have been identified.

Group I—(*a*) Marked anæmia, count often less than 1 million RBC's—macrocytic hyper- or orthochromic in type. (*b*) Marked diarrhœa. (*c*) Little or no glossitis.

Group II—(*a*) Moderate anæmia, count between 2·5 - 3 million RBC's. (b) Marked glossitis. (*c*) Diarrhœa variable, often relapsing in character.

Group III—(*a*) Slight anæmia, count often above 3·5 million RBC's. (*b*) Marked glossitis. (*c*) Marked diarrhœa.

Group IV—(*a*) Slight anæmia, RBC count in the neighbourhood of 4 million. (*b*) No diarrhœa. (*c*) Marked œdema of ankles. (*d*) Absent ankle and knee jerks. (*e*) Bradycardia. (*f*) Glossitis may or may not be present.

Group V—(*a*) No anæmia. (*b*) No diarrhœa. (*c*) Marked glossitis.

The following is the analysis of the cases according to the above heads:

Group	*No. of Cases*
I.	151(30·2%)
II.	203(40·5%)
III.	81(16·2%)
IV.	6(1·1%)
V.	59(12 %)

Short Description of the Actual Symptoms

Stool—In majority of cases (75 per cent) stools were pale, watery in character often alternating with periods of constipation and sometimes of stools normal in colour and consistency for a period varying from 2-5 days. The fat content was normal in about 80 per cent of the cases. It is only in the very late stage of the disease, that the stools were bulky, offensive, greasy and frothy in character with excess of hydrolysed fat. The average figure in such cases varied from 35-40 per cent of the total fat content.

Steatorrhœa had arisen *de novo* in about 18 per cent of cases; and as a sequel to some form of ill-defined dysenteric symptoms in the rest. It is interesting to compare these figures with these of Manson Bahr who had shown that 40·5 per cent of his 200 sprue patients gave a history of intestinal disorder.

Further while serving in the Middle East, cases of chronic and relapsing dysentery of varied aetiology have been seen in the course of which stomatitis and steatorrhœa developed; many of whom in addition to inadequately treated dysentery gave a history of inadequate diet.

This, of course, adds an additional evidence that steatorrhœa probably is the result of vitamin deficiency and links sprue syndrome at least in part as due to deficiency of vitamins.

Anæmia—The degree of anæmia and type are classified according to M.C.V. and M.C.H.C. The normal range of M.C.V. was calculated on the basis of the normal range being 75-96 cubic microns and a normal range of M.C.H.C. to vary from 28-34 per cent.

The following is the analysis of 500 cases:

Cause	*Type*	*No. of Cases*
Vit. B.	Normocytic orthochromic ..	15(3·1%)
Iron.	Normocytic hypochromic ..	11(2·2%)
do.	Macrocytic hypochromic ..	57(11·3%)
Dimorphic.	Macrocytic orthochromic ..	236(47·3%)
do.	Microcytic orthochromic ..	5(1 %)
do.	Microcytic hypochromic ..	27(5·3%)
do.	Macrocytic hyperchromic ..	149(29·8%)

Some interesting points emerged in the investigation of anæmias:—

(*i*) Majority of cases in the group of macrocytic orthochromic anæmia gave a history of malaria.

(*ii*) Out of 57 cases of macrocytic hypochromic anæmia, 15 had a very heavy infection of hookworm and it seemed probable that this infection was responsible for iron deficiency and hypochromia.

(*iii*) The cases of normocytic hypochromic anæmia showed dual deficiency. Iron deficiency was usually the major deficiency and the blood count improved considerably by iron alone, but by giving diet rich in protein, vitamin B and liver extract recovery was more speedy.

(*iv*) No suggestive confirmation of this dual deficiency was obtained by examination of bone marrow due to lack of time and the necessary facilities.

Glossitis—Tongue changes are variable and often depends as to which of the deficiencies is more predominant. In riboflavin deficiency, in the early stages there is stripping of the fur and the epithelium of the

edges and tip, most marked at the tip leaving these areas smooth, dark red magenta in colour, with flattened papillæ. The centre of the tongue in such cases is usually covered with thick white fur. As the case advances the tongue is completely stripped of fur and is smooth but not glossy and slightly swollen with lateral indentations caused by teeth. Longitudinal fissuring usually occurs, although at the edges there may be a few transverse fissures. Soreness is complained of.

In nicotinic acid deficiency the tongue is glazed and scarlet in colour with rather shallow longitudinal fissures.

It is more common to see a combination of these deficiencies. Glossitis may also be associated with angular stomatitis and cheliosis. This may extend backwards into the pharynx giving rise to dysphagia. In three of my cases the picture was one of Plummer-Vinson syndrome.

Prognosis

Mortality has been fortunately very low. Out of a total 1257 cases treated under my care 15 died eventually giving us a figure of 1·5 per cent.

It has been brought home to us that given adequate treatment started early in the course of the disease and for a sufficient period, the majority of these cases will make full recoveries. Such cases as we have been able to follow have shown no tendency to relapse after discharge from the Hospital. We feel therefore that the indiscriminate use of the term sprue is to be deprecated in view of the implication that the diagnosis conveys to the lay mind.

Treatment

(*i*) Rest.

(*ii*) Diet—Few principles in selecting a diet are:

(*a*) The diet should be of high caloric value (at least 2500 calories) with full vitamin content in easily assimilable form. The chart of diet has been framed by me and which has been used in these cases. It has proved very satisfactory. It permits great variation in attainment of high calories, high protein, low fat, low carbohydrate and high vitamin intake. It is adapted for fluids, semi-solid or solid diets arranged in such a way as to suit the tastes of an Indian; its items are all within Standard Hospital Ration Scales (*vide infra*).

(*b*) It should be noted that these cases are liable to develop diarrhœa if put too soon on a full diet. The intestinal mucosa in malnutrition is often œdematous and even when it is not, it is reasonable to assume that the gastrointestinal tract needs re-education in the digestion of normal diet. In extreme cases body reserves may be so exhausted that not enough are left to supply even the enzymes necessary for digestion. Another aim in such cases is to restore the depleted body tissues. It is therefore essential that the change of diet from one to another should be very slow.

(*iii*) Symptomatic—(*i*) *Diarrhœa* very often is controllable with proper dietetic restrictions. If still persistent—controllable by simple demulcents as ispaghula. In very severe and intractable cases sulphaguanidine will invariably control it though in some cases it may even recur until such time as the resistance of the patient and the depleted body tissues are restored as a result of repeated blood transfusions.

(*ii*) *Glossitis*—Nicotinic acid, riboflavin or yeast by mouth are useless in the beginning of the treatment when the patient is unable to absorb them as a result of his intestinal disorder. Parenteral use of these drugs is advocated if supplies are available.

Blood Transfusions—Has been the sheet anchor of treatment in my cases. It should be given slowly, in small quantities (500 c.c.) and repeated very frequently (after 2-3 days). Until such times as the blood picture is raised well above 3·5 million RBC. and maintained there for 10-15 days or the diarrhœa is controlled.

The blood replaces not only the red cells which are deficient in these cases, but also the proteins and vitamins. It gives an extra stimulus to the hæmopoietic system to regenerate the red cells.

The earlier this line of treatment is taken up and maintained, the better would be the results. Isolated small transfusions are useless.

It should be remembered that the first blood count is often higher than what it actually is, due to hæmoconcentration.

(*b*) Plasma although not regarded as an ideal substance for parenteral nutrition has proved very satisfactory in 6 fairly severe cases of malnutrition group III (with little anæmia) and may well be used temporarily when supplies of protein hydrolysates are not available, in cases with low plasma proteins.

(*c*) I have no experience of protein hydrolysates although I do feel that they should be very satisfactory in some of these nutritional disorders without anæmia.

The use of liver extract is only advocated in the later stages of treatment when macrocytosis still persists. Given in earlier phases it does not improve the blood picture of these patients.

Basic Ration Scale (I.T.)

Food	*Portion*	*Calories*	*Proteins in grams*	*Vitamin A & D*	*Vitamin B Complex*	*Vitamin C*	*Issues*
Atta ..	12 oz.	2,400	72	..	+	..	Daily
Rice ..	12 oz.	2,400	24	..	..	..	"
Dal ..	4½oz.	450	..	..	+	..	"
Ghee ..	2¾oz.	605	..	+	..	..	"
Milk Fresh ..	6 oz.	112	2	+	+	..	"
Potatoes fresh ..	6 oz.	150	..	..	..	+	"
Sugar ..	2¼oz.	247	..	..	..	..	"
Fruits fresh ..	4 oz.	..	..	..	..	++	"
Total	..	3,964	74/26	2	3	3	"

Total Diet	*Calories*	*Proteins*	*Vitamin A & D*	*Vitamin B Complex*	*Vitamin C*
Atta eater ..	3,964	74	++=2	+++=3	+++=3
Rice eater ..	3,964	26	++=2	+++=3	+++=3

FIELD SERVICE SCALE (I.T.)

Food	Portion	Calories	Proteins in gms.	Vitamin A & D	Vitamin B Complex	Vitamin C	Issues
Atta	24 oz.	2,400	72	..	..	..	Daily
Rice	12 oz.	2,400	24	..	..	..	„
Dal	3½oz.	375	24	..	+	..	„
Ghee	2¾oz.	605	..	+	..	..	„
Potatoes	4 oz.	100	..	..	..	+	„
Sugar	2½oz.	275	..	..	..	..	„
Meat fresh lean	2 oz.	80	10	..	..	..	„
Fruits fresh	8 oz.	..	..	..	..	++	Thrice weekly
Total	..	3,835	106/58	1	1	3	

Total Diet	Calories	Proteins	Vitamin A & D	Vitamin B Complex	Vitamin C
Atta meat eaters	3,835	106	+=1	+=1	++=3
Rice meat eaters	3,835	58	+=1	+=1	++=3
Atta veg. eaters	3,755	96	+=1	+=1	++=3
Rice veg. eaters	3,755	48	+=1	+=1	++=3

The following is a list of a well-balanced diet as adapted from the scheme drawn by the League of Nations:

2600 calories corresponding to average adult daily requirements.

Raw milled rice	10 ozs.
Cambu	5 „
Milk	8 „
Pulse (dal arhar 1 oz.) (black gram 2 oz.)	3 „
Non-leafy Veg:	
Bringal	2 „
Ladies fingers	1 „
Snake gourd	1 „
Cluster beans	1 „
Drum stick	1 „
Leafy Veg:	
Amaranath leaves	2 „
Drum stick leaves	1 „
Spinach	1 „
Gingelly oil	
Fruit:	
Mangoes	1 „
Ripe plantains	1 „

DIET CHARTS

Clinical Picture	Diet 'A'	Quantity in Oz.	Calories	Protein in gms.	Vitamin A & D	Vitamin B Complex	Vitamin C	How Administered
1. Very acutely ill.	1. Milk	60	1125	56	++	+	..	Milk or Curd 8, 11, 15, 20 hrs.
2. Diarrhœa 8-10 stools a day, watery, semi-solid or frothy.	2. Chicken soup	8	240	40	..	..	..	Chicken Soup 12 hrs.
3. Glossitis—red, washed, glazed, angry looking ulcers present.	3. Sugar with Lime Sugar Lime	3 3	345	..	..	..	++	Fruit Juice—3 p.m.
	4. Liver	2	80	10	+++	+	..	
	5. Biscuits	2	140	..	..	..	..	Biscuits—4 p.m.
4. Flatulent distension of abdomen with tympanitis.	6. Fruit juice	4	..	..	..	..	+	
5. Hb. below 30%	7. Marmite	½ B.D.	..	..	..	++	..	Marmite—9 a.m. & 5 p.m.
	Total		1930	106	5	4	3	

Clinical Picture	Diet 'B'	Quantity in Oz.	Calories	Protein in gms.	Vitamin A & D	Vitamin B Complex	Vitamin C	How Administered
1. Moderately ill.	1. Milk	60	1125	56	++	+	..	Milk (as pure or in curd form) 6 a.m., 8 a.m., 10 a.m., 12 a.m., 4 p.m. & 8 p.m.
2. Diarrhœa 5-7 stools.	2. Chicken broth	3	240	40	(Two extra eggs or 1 oz. butter in case of vegetarians).			
3. Glossitis—red, glazed or smooth.	3. Eggs 2		120	8	+	..	..	Biscuits 4 p.m.
4. Tympanitis after food only.	4. Liver	4	160	20	+++	+	..	Marmite 9 a.m. & 5 p.m.
	5. Sago with Milk	6	150	..	..	..	..	Sago during day.
	6. Kichri, Rice wt. raw ½oz. Dal	2	164	2	..	..	..	Kichri—at night.
5. Hb below 50%	7. Biscuits	2	140	..	..	..	..	
	8. Lime & Sugar Sugar Lime	3 3	345	..	..	..	++	
	9. Marmite	½ B.D.	..	..	..	++	..	
	Total		2444	126	6	4	2	

DIET CHARTS (CONTINUED)

Clinical Picture	*Diet 'C'*	*Quantity in Oz.*	*Calories*	*Protein in gms.*	*Vitamin A & D*	*Vitamin B Complex*	*Vitamin C*	*How Administered*
1. Diarrhœa upto 3 stools, semi-solid, or well formed.	1. Milk	60	1125	56	++	+	..	Milk same as above (4 hourly feeds).
	2. Chicken (Cooked without spice	1 lb.	480	80	(Two extra eggs or 1 oz. butter in case of vegetarians)			
2. Glossitis still present but epithelium and papillæ regenerating.	3. Eggs 2		120	8	+	..	..	Curd & Kichri—Day.
	4. Liver	4	160	20	+++	+	..	Bread & Chicken Soup at night.
	5. Kichri, Rice wt. raw	3½						
	Dal wt. raw	1½	500	..	..	..	..	
4. Little or no tympanitis.	6. Bread	4	280	..	..	..	..	Rest as above.
	7. Biscuits	2	140	..	..	..	..	(*Note.* Kichri is cooked with quantity of rice and dal in Col. 2 with water and a little salt to taste).
5. Hb between 50-60%	8. Sugar & lime							
	Sugar	3						
	Limes	3	345	..	..	..	++	
	9. Jelly	4	50	..	..	..	..	
	10. Marmite	1	..	..	..	+++	..	
	Total		3200	164	6	5	2	

Diet D which is really a Modified 'Convalescent Diet' + 16 ozs. extra milk and chicken cooked without spice

Clinical Picture	*Diet 'D'*	*Quantity in Oz.*	*Calories*	*Protein in gms.*	*Vitamin A & D*	*Vitamin B Complex*	*Vitamin C*	*How Administered*
1. Patient having 1-2 stools.	1. Atta (Flour)	16	1600	48	..	++	..	
2. Well formed, non-offensive stools without any gas bubbles and normal fat contents.	2. Dal (Wt. raw)	2	160	10	..	+	..	
	3. Ghee	2	200	..	+	..	..	
	4. Mutton	4	160	20	..	..	..	Times as in Diet 'C'.
	5. Sugar	3	345	..	..	..	..	Morning meal (Dal cooked in 1 oz. ghee with salt & onions & eaten with 8 ozs. atta made into chapatis.
3. No tympanitis and the patient having been free for at least 3 weeks.	6. Milk	36	620	30	++	..	..	
	7. Eggs 2		120	8	+	..	..	
	8. Marmite	½	..	..	..	++	..	
	9. Lime juice	BD	..	..	..	..	++	Evening meal—(Dal cooked in 1 oz. of ghee with salt and onions and pepper to taste, eaten with 8 ozs. atta made into chapatis.
4. Hb over 60% with little or no glossitis.	10. Chickens	1 lb.	480	80	..	..	..	
	Total		3675	196	4	5	2	

N. B. In cases of vegetarians in

(*a*) Diet 'A' substitute 'Two extra eggs or one ounce of butter for chicken broth'.
(*b*) Diet 'B' substitute 'Two extra eggs or one ounce of butter for chicken broth'.
(*c*) Diet 'D' substitute 'Vegetable curry (made of vegetable available in season like peas, cauliflower, or egg curry made of two eggs in lieu of mutton. If vegetables of season are poor in proteins in addition to vegetable give 10 ozs. of milk feed at night.

If vegetables of season are poor in proteins in addition to vegetable give 10 ozs. of milk feed at night.

(*d*) In diet 'B', 'C' & 'D'—milk is given as pure milk, or curd 8 ozs. in the morning. or in the form of *khir* (milk 8 ozs. with rice 1½ ozs.) and milk & eggs, either egg flip or egg custard.

INVESTIGATION OF THE SO-CALLED PRIMARY ABDOMINAL TUBERCULOSIS

A Preliminery Report

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For many years, one has had the feeling that the diagnosis of primary abdominal tuberculosis is being made much too frequently and without a critical appraisal of the various other possibilities, possibly because an emphatic and certain rebuttal of such a diagnosis is almost impossible by the various diagnostic aids at our disposal; the contrast from pulmonary tuberculosis, where careful and repeated skiagraphy and sputum examination can make the diagnosis certain, is marked. During the last seven years I have had occasions to come in contact with leading physicians all over the country and I have been surprised to find almost all of them holding the same view *i.e.*, primary abdominal tuberculosis is very common.

SYMPTOMS

The symptom-complex on which this diagnosis is based is somewhat as follows:—

Discomfort or dull pain in the abdomen exacerbated at times after food, sense of fullness in abdomen, irregular bowels or so-called alternate diarrhœa and constipation, anorexia, anæmia, loss of weight, occasionally slight temperature, and not infrequently chronic glossitis. On examination the only positive signs are anæmia and vague tenderness all over the abdomen, specially around the umbilicus or the right iliac fossa. No masses are palpable, neither is the abdomen doughy, except in a few cases. The lungs are perfectly normal and there are no foci of tuberculosis in any part of the body. More females than males show this symptom-complex and the age is usually from 15 to 45. The past history is seldom of importance, except that of attacks of dysentery off and on. Many of these improve with a few injections of calcium and use of cod liver oil etc. and the absurd claim of having cured abdominal tuberculosis is made. Knowing, as we do, what an inveterate foe tuberculosis in every form is, it is impossible to agree that these were cases of tuberculosis.

CLASSIFICATION

It will be interesting to know what the various authorities say about abdominal tuberculosis. A careful and painstaking survey of available literature is remarkable for its paucity. Most writers divide abdominal tuberculosis into 3 groups:—

(*a*) Intestinal, (*b*) Glandular, (*c*) Peritoneal—and further subdivide these three into primary and secondary.

Intestinal Tuberculosis—All writers are agreed that the vast majority of cases of intestinal tuberculosis are secondary to frank lung disease. Brown and Sampson (1930) say that about 50 per cent of all cases of pulmonary tuberculosis show intestinal involvement at autopsy. Osler and McCrea (1935) quote the Munich figures at 56 per cent. Maxwell (1936) states it below 30 per cent but adds "Post-mortem statistics are such a hazardous guide". Tribedi (1941) and Ukil (1942) give 41 per cent, while Boyd (1944) goes upto 80 per cent. From personal experience one would give a much higher figure—it would seem that almost all cases of advanced pulmonary tuberculosis and the majority of early cases show definite intestinal involvement. So much for secondary intestinal tuberculosis.

As regards primary abdominal tuberculosis, almost all are unanimous that it is very uncommon. Brown and Sampson (1930), Osler and McCrea (1930), Goldberg (1935), Amberson Junior (1936), Shrikhande (1936), Maxwell (1936), Conybeare (1937), Hurst (1944) and Boyd (1944) repeat the same statement. Tribedi (1941) in his valuable autopsy statistics says that only 5 per cent of all cases of intestinal tuberculosis are primary. Of the two major types of intestinal tuberculosis, the ulcerative and the hyperplastic, the latter is supposed to be almost always primary. In Tribedi's 10 cases of primary tuberculosis (5 per cent of the total), as many as four belong to this type, which, according to Hurst (1944) are not really tuberculous at all but should be included in the group of regional ileitis. This leaves us with only 3 per cent. His reports are obviously from the very poorest strata of society, and it would be not unreasonable to expect much lower figures in actual practice.

The symptomatology of secondary ulcerative type is somewhat similar to the one given above, except that diarrhœa is a constant feature when colon is predominantly involved, while constipation and/or diarrhœa is observed when the small intestine is the chief site of involvement. The stool examination for tuberculosis may be positive, occult blood is frequently present, and the Triboulet test may be positive. A barium meal examination shows hypermotility, irregular filling defects and spasm with tenderness of the intestines. This similarity probably accounts for the frequency with which primary abdominal tuberculosis is diagnosed, because it is forgotten that several other conditions can produce a similar symptom-complex.

Abdominal Glandular Tuberculosis—The chief groups of glands affected are the mesenteric and the para-arotic ones, but their primary involvement in this region is very much more uncommon except in young children, than the involvement of the cervical and the mediastinal groups. Their involvement secondarily to intestinal tuberculosis is, however, common. In such cases, palpation shows definite irregular adherent masses, which are tender.

Peritoneal Tuberculosis—There are three types—ascitic, loculated and fibrocaseous. The ascitic is the acute or subacute type with low fever (which is, sometimes, surprisingly absent) with a tubercle-studded peritoneum, and signs of free fluid and generalised abdominal tenderness; the loculated variety follows the same pattern but usually a fluctuating mass is palpable. In the last variety, we may get a palpable, rolled omentum or adherent and palpable intestinal masses with alternate diarrhœa and constipation or

chronic intestinal obstruction and fæcal fistula formation, and palpation gives a "doughy" feel while the spleen and liver are often palpable. Here also, the primary variety is very uncommon. In the great majority it is secondary to frank intestinal, glandular or fallopian tube infection. It is in this type that opening of the abdomen or a pneumoperitoneum produces good results.

Differential Diagnosis

If a critical analysis of the above facts is kept in mind, it becomes clear that we should hesitate very much before diagnosing primary abdominal tuberculosis. The other conditions to be constantly kept in mind are the following:—

1. *Mucous or catarrhal colitis*—usually an after-effect of badly treated bacillary dysentry—a disease almost universal in this country because of primitive sanitation.
2. *Amœbic colitis*—in these stool examination may show cysts of Entamœba histolytica.
3. *Non-specific enterocolitis*—often following frequent and injudicious use of purgatives at the slightest provocation.
4. *Ulcerative colitis* with passage of almost pure pus with great loss of weight and anæmia etc.
5. *Spastic colitis*—an unsatisfactory term for cases of colitis in which frequent spasms with colicky attacks are a common feature.
6. *Chronic appendicitis*—a diagnosis much favoured by surgeons.
7. *Malignancy* with alternate diarrhœa and constipation and loss of weight.
8. *Chronic fallopian tube and ovarian infection* —to be looked out for by a careful vaginal examination.
9. *Regional ileitis*—a not very uncommon condition with a palpable mass in the right iliac fossa and suggestive x-ray picture. So-called hyperplastic tuberculosis must be included in this group—Hurst (1944).
10. *Sprue and cœliac disease* associated with fatty diarrhœa, anæmia, loss of weight.
11. *Pellagrinous diarrhœa* should also be kept in mind.
12. *Other causes* of diarrhœa associated with loss of weight and anæmia *e.g.,* adrenal disease etc.

The point I wish to stress is that, unless the above classical description of abdominal tuberculosis based on careful pathological work, is grossly incorrect, the great majority of cases that are diagnosed as primary abdominal tuberculosis are being wrongly diagnosed and therefore wrongly treated. Moreover, the psychological trauma on the patients' mind when he is wrongly told that he is suffering from tuberculosis must be great. It is upto us, in the interests of scientific exactitude, to be more careful about our diagnoses.

The alternative diagnosis, to my mind, appears to be a colitis either after an attack or attacks of bacillary dysentery which is so universal in this country and which is as universally badly treated, or chronic amœbic dysentery or a non-specific 'chemical' colitis due to prolonged misuse of purgatives. To prove this, the following investigations have very recently been initiated in the cases under my charge in the K. G. Hospital, Lucknow. In addition to the usual history, the following points are specially asked for:—Past history of dysentery with its clinical manifestations and the treatment adopted, history of prolonged contact with an open case of pulmonary tuberculosis, and the use of purgatives. In the clinical examination, in addition to the usual systematic examination, careful abdominal and rectal examination, and also vaginal examination in the case of women.

The stools are examined for T.B. by the antiformin method and the Triboulet's test is done in addition to the usual microscopic examination. Skiagraphy of the chest and of the gastrointestinal tract with barium meal and barium enema are done. The tuberculin test (Mantoux) is also performed. Agglutination test against the dysentery group of organisms is done, total R.B.C., Hb. per cent and total and differential W.B.C. count are done. Fractional test-meal is also done.

Treatment

In these cases, I have been following the undermentioned technique of treatment:—

1. Rest in bed, at least in the early stages.
2. Warmth, particularly to the abdomen in the form of a warm binder. A bland ointment locally exerts a certain amount of psychological effect.
3. If a specific cause is found *e.g.,* cysts of Ent. histolytica, it is appropriately treated.
4. If diarrhœa is a prominent feature, sulphaguanidine—4 tablets every 4 hours, along with an acid mixture (as many cases are suspected to have achlorhydria) usually controls it. Injections of calcium gluconate 10 per cent 10 c.c. intravenously daily are helpful. If constipation is a feature, two teaspoonfuls of ispaghula husks may be given twice a day.
5. Diet is ample (most of these patients are very under-weight) but bland, with as little roughage as possible, so that intestinal irritation is avoided. These people do not usually tolerate milk well, because it tends to produce flatulence. Fresh cow-milk-curds is extremely valuable, as I can testify from personal experience. Replacement of the morning coffee or tea with fresh curds is particularly insisted upon. It can be taken sweet or salted, according to taste. All spices, pickles, green vegetables, tough meat highly seasoned and greasy dishes are particularly avoided. Fresh fruit juice should always be taken.
6. The existing anæmia is treated in the usual way with suitable anti-anæmic drugs. Purgatives are rigorously avoided. Vitamins in good doses are always given—particularly the vitamin B-complex which plays such an important part in the health of the oro-gastrointestinal mucosa. In cases with chronic glossitis, nicotinic acid 100 mgms. thrice a day are given. Belladonna in some form is given if spasms are present. It is also proposed to try sulphaguanidine in large doses orally to disinfect the intestinal tract.
7. The daily use of isotonic low pressure enemata forms the basis of this treatment. Two drams

of sodium chloride and one dram of sodium bicarbonate are dissolved in two pints of warm water (this giving an isotonic solution) and the enema is taken after the morning bowel evacuation (in those accustomed to it). It is best taken while lying in bed and the enema-can is put at a height of never more than 8" above the anus. The fluid takes about 10-15 minutes to go in, this is retained for 30-60 minutes and then evacuated. This 'low-pressure' is essential because high pressure gives rise to irritation and active peristalsis of the bowels leading to quick rejection of the fluid, thus defeating the very object of treatment, *viz.*, gentle washing of the inflamed bowel. The sodium bicarbonate helps to dissolve the mucus. In cases with much inflammation as shown by the microscopic examination, I usually add 4-6 tablets of enterovioform which acts as a sedative and antiseptic. This treatment is based on the analogy of treatment of chronic inflammation in any part of the body. The enema can be taken at any time of the day or night, convenient to the patient—as the whole process takes up an hour or more.

8. The psychological factor should be constantly kept in mind. These patients have almost always been told that they have intestinal tuberculosis. They should be reassured; a brief explanation of the pathological process and of the rationale of the treatment will always bring forth from the intelligent patient an active and willing co-operation. They should be warned against expecting miracles. The possibility of occasional relapses should be told to them, otherwise they are apt to become unduly pessimistic at their appearance.

That one does come across genuine cases of definite primary abdominal tuberculosis is undoubted, but these must be classifiable in the above scheme.

Case Reports

Case 1—S. M. (40 years) had been prone to occasional attacks of intestinal colic. History of severe bacillary dysentery in childhood. These attacks of pain became very frequent and severe in 1926 and were associated with several semi-solid stools daily, loss of weight, slight temperature, anæmia and vague tenderness all over the abdomen but specially round the umbilicus and the right side. As the patient was a student in a Medical College, the various physicians examined him and unanimously pronounced it as a case of abdominal tuberculosis. The chief surgeon said it was chronic appendicitis, and operated upon him. On the operation table, however, there was absolutely no sign of any tuberculosis, the appendix was normal but was removed, and the wound was stitched up. After a couple of months, the old trouble restarted. This was gradually controlled with the above regime and the person has regained good health, and since then he has had only two mild attacks in 1930 and 1943.

Case 2—J. J. (38 years) gave almost exactly the same history and sequence of events. Laparotomy showed no abnormality and the appendix was removed. A few months later, trouble restarted and was gradually relieved of the trouble when placed on the above regime. Hhas been all right for over 5 years now.

Case 3—B. K. (20 years) seen five years ago with extreme loss of weight, severe anæmia, vague tenderness all over the abdomen, stools very frequent—15 to 20 times a day with pure pus, a skiagraph after barium enema showed loss of haustration. Diagnosed intestinal tuberculosis outside but here the diagnosis was ulcerative colitis. Bland liquid diet in the form of curds and white bread started, the macrocytic anæmia was treated with acid and iron mixture, with injections of liver and vitamin B-complex. Low pressure enemata were given daily. Slowly the patient started improving and was all right in about 3 months' time. Last seen, a week ago and found perfectly normal.

Case 4—Mrs. T. (25 years). History of gradual onset after childbirth with frequent attacks of abdominal pain, tenderness round the umbilicus and right iliac fossa, irregular stools, loss of weight and anæmia. P. V. examination negative, stool examination was negative, except for pus cells. Diagnosed and treated for abdominal tuberculosis for over one year with little improvement. Gradually improved with the above regime and was cured in about 4 months. Four years have now passed without any trouble.

Case 5—Mrs. B. (30 years) admitted into hospital on 17-11-45 with low fever, attacks of abdominal pain with tenderness round the umbilicus, and loss of weight. Stool examination was completely negative and barium enema showed an unduly dilated pelvic colon which was at times visibly distended with flatus. P. V. showed non-tubercular chronic erosion of the cervix while the tubes and ovaries were normal. Improving with the usual regime.

Case 6—B. C. (38 years). Loose stools with griping pain, marked loss of appetite, severe anæmia, troublesome atrophic glossitis with sore angles of mouth. Diagnosed as abdominal tuberculosis and treated for 2 months without benefit. Stools showed large number of pus cells. Bland diet with acid and iron mixture and nicotinic acid with riboflavin injections produced considerable improvement and the patient seems to be on the way to recovery.

These very imperfect records have been described only to stimulate further study. Regular work on the subject has only just been started and I hope to present a much more comprehensive and methodical report on this subject.

Summary

A symptom-complex, which is almost universally diagnosed as primary abdominal tuberculosis is described. Reasons are given which militate against such a facile but incorrect diagnosis. An alternative diagnosis is suggested, a few cases are described and the suggested treatment is detailed. It is emphasized that this is a preliminary note, submitted to stimulate discussion and suggestions.

Acknowledgments

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(*Continued at foot of page 328*)

SURGICAL PROBLEMS IN THYROID DISEASE*

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Thyroid, the shield gland of the body working under the direction of its master, the anterior pituitary (a functioning anterior lobe is essential to the normal development and function of thyroid), through its thyrotropic hormone is not only concerned with the hormone production but also regulates the iodine metabolism of the body. It differs from all other organs and tissues not only in its high concentration of iodine (containing about 1/5th of all the iodine in the body) but also in its affinity for iodine. It is the only storage depot for iodine, rapidly traping every bit (of iodine) entering the circulation.

Thyroid hormone (thyro-globulin=thyroxine+ di-iodotyrosin) plays a far reaching role in the economy of the body. It has a lot to do with the rate of growth, the differentiation of tissues, with the rate of energy exchange, with metabolism of proteins carbohydrates, fats, lipoids, salts, and water; with the irritability of the various tissues and with activity of other endorcrines. The hormone acts as a catalyst and also exerts some influence on the permeability of the cell membranes. All the endocrine glands are normally in a state of hormonic balance so that when the function of one of them becomes deranged changes often occur in some of the others which are thought to be compensatory in nature although it must not be assumed that this is a direct quantitative compensation. Thus when the thyroid secretion is deficient, hypertrophic changes have been noted in the pituitary and pineal glands, while the sex glands undergo retrogressive changes. The hormonic antagonism between the thyroid and pancreas is well proved.

Development—The thyroid is developed from three separate buds, a central and two lateral. The central bud appears as a diverticulum from the floor of the mouth between the three elevations from which the tongue is developed. It grows downwards as a solid column of cells, passing through the developing hyoid bone, and bifurcates to form the isthmus of the gland and the major portion of the lateral lobes. The lateral buds arise from the fourth branchial cleft as thick walled saccular glands. They correspond to the post branchial bodies found in most vertebrates; the amount of human thyroid developed from the lateral buds is doubtful, some believe that they degenerate to mere vesicles in the lateral lobes. The stalk connecting the embryonic thyroid with the back of the tongue becomes constricted to form the thyroglossal duct which at birth is represented by a dimple called foramen cæcum.

The compound origin of the gland is indicated by its blood supply, the medial portion being mainly supplied by the superior thyroid artery, while the lateral portions are supplied from a branch of the fourth aortic arch, the inferior thyroid. In structure the primitive gland consist of masses of solid column of cells, and it is only later (fourth fœtal month) that these become hollowed out and divided by the accumulation of colloid to form vesicles.

During the course of development portions of gland may become displaced. A nodule occurring at the base of the tongue is called lingual thyroid and forms the commonest abnormality. Here thyroid gland gives rise to a rounded swelling at the back of the tongue beneath the foramen cæcum. Removal of a lingual goitre is often followed by myxœdema. It has therefore been recommended that some portion of the excised gland should be grafted into the medulla of the tibia. Portions detached during the process of descent may form accessory thyroid in the neck *e.g.*, there may be an extra lobe near the angle of the jaw or the thyroid tissue may be scattered all along the thyroglossal tract or a bit detached into the superior mediastinum may later on give rise to one form of retrosternal goitre.

The gland is composed of two lateral lobes connected by an isthmus. Each lateral lobe extends vertically from the middle of the side of the thyroid cartilage to the sixth ring of the trachea, the isthmus covering the second, third and fourth. A triangular projection of the gland tissue called the pyramidal lobe extends upwards from the left side of the isthmus, and is connected to the hyoid bone above by a fibrous band of a muscle slip called levator glandulæ thyroideæ.

The anterior surface is covered by the infrahyoid or ribbon muscles, the strenomastoid overlapping.

The medial surface is related to two tubes, œsophagus and trachea; two nerves—recurrent and external laryngeal; two muscles—inferior constrictor and cricothyroid.

The posterior surface overlaps the common carotid artery and covers the terminal part of the inferior thyroid artery.

Function—The function of the thyroid consists mainly in the production of several hormones. They enter the blood stream by reabsorption from the colloid matter in the acini. One of these hormones is known as thyroxine and is available also in synthetic form. It is conspicuous for its iodine content, which amounts to as much as 60 per cent. The thyroid also produces other iodine containing substances. They are thyroglobulin, iodothyrin and di-iodotyrosin. While most of the investigations in recent years have centred on the nature, properties, and effects of thyroxin, the importance of di-iodotyrosin has been stressed only recently. This substance is the biological antagonist to thyroxin, because the effects of thyroxin can be counteracted by simultaneous administration of di-iodotyrosin.

The iodine content of the normal human thyroid is between 6 and 7 milligrams. It varies, however, with the different races and depends also upon exogenous factors such as climate, season and nutrition. It is higher during the summer and lower in winter. Proteinrich diet, and particularly meat, increases not only the iodine content but also the size of the thyroid in the experimental animals. Vegetarian diet has an opposite effect. During the growth period thyroxin exerts an obscure controlling influence so that in its

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absence cretinism follows. In this condition there is deficient growth not only of bones but also of the soft parts, the brain and the sex glands. In the adult deprived of thyroid secretion a similar set of changes occur, although the actual stature cannot, of course, be affected. Slowness of cerebration, and of movement, myxomatous hypertrophy of subcutaneous tissues and trophic change in the skin and glands produce a typical syndrome called myxœdema. Excess of secretion, on the other hand, produces an opposite set of symptoms—the pulse is accelerated, the skin hyperactive, cerebration rapid and the whole nervous system in a state of over excitability; the sex functions also are deranged. Metabolism occurs at an increased rate which sometimes exceeds assimilation and leads to wasting.

The control which the thyroid exerts over metabolism can be estimated by examining the basal metabolic rate. In brief, by analysis of the inspired and expired air with regard to oxygen intake and CO_2 output, it is possible to deduce the rate at which the ultimate oxygenation and break down of all the materials in the tissues is occurring—a rate which is found to vary *pari passu* with the activity of the thyroid. This method is now largely used as a means of testing the relative values of the various methods of treatment.

The function of the thyroid can be substituted for by implantation of thyroid tissue or by medication; the thyroid hormone is not destroyed by digestive juices and is applicable orally. The effects of gland, gland extract, pure and even synthetic thyroxin are essentially identical. The result of such administration becomes manifest by an increased metabolic activity.

Each thyroid cell is under sympathetic nerve control through a terminal reticulum. If impulses from the central nervous symtem are abnormally strong, no matter through what cause, a hyper- or dysfunction ensues. Under pathological conditions the terminal reticulum is destroyed and the thyroid is out of control.

Marine asserts that there exists a specific thyroid nerve made up of fibres from the superior cervical ganglion of the sympathetic and parasympathetic fibres come from the superior laryngeal branch of the vagus.

Utilization of the Basal Metabolic Rate—The ever increasing employment of determination of the basal metabolic rate to confirm or disprove diagnosis of thyroid disease, to control the pre-operative treatment of hyperthyroid patients and to adjust the thyroid dosage of those exhibiting a hypothyroid condition, renders the precise evaluation of this test, a matter of considerable importance. In fact B.M.R. is the means of measuring quantitatively the level of thyroid function. Apart from thyrotoxicosis, B.M.R. is raised in fever, acromegaly, leukæmia and severe anæmias.

The Chinese are stated to exhibit a lower metabolic rate than the Western races so also are Eastern people. Eskimos give values averaging 33 per cent higher than the standards.

While the precise causes of these racial differences cannot be stated, differences in diet are undoubtedly an important factor. Thus the low figures obtained for medical students in Madras (12 per cent for males; 16 per cent for the females) have been attributed to low protein diet and ready muscular relaxation.

Under-nutrition markedly affects the B.M.R. Abnormally low diet can depress the rate more than 20 per cent. Protein free diet will produce a rapid fall in the basal metabolic rate of a normal individual within a few days. During pregnancy there is a slow rise. Levels and trends of metabolic rates are more significant than single absolute values.

CLASSIFICATION OF DISEASES OF THYROID

I.	Deficient secretion	..	Cretinism Myxœdoma
II.	Simple goitre	..	Colloid goitre of puberty 23·3% Adeno-parenchymatous goitre 32·5%
III.	Thyrotoxic goitre	..	Primary Graves' disease 32·3% Secondary Graves' disease
IV.	Neoplasms ..	..	Benign Malignant 1·1%
V.	Inflammations	..	Acute Chronic.

COLLOID GOITRE OF PUBERTY OR ADOLESCENT GOITRE—It is usually a simple and transient affection. The main symptom is swelling in the neck usually of a horse-shoe shape. Girls are more frequently affected than boys at about the age of 14 to 18. The thyroid is evenly enlarged and soft to touch. Usually the enlargement gradually subsides and has disappeared by 20-22 years.

Morbid anatomy—There is hyperplasia of both the secreting and fibrous elements of gland so that gland becomes enlarged as a whole. This hypertrophy is in some way connected with physiological change of puberty, pregnancy, lactation and climacteric. It is of surgical interest for two reasons:—(1) It occasionally persists and gives rise to thyrotoxic symptoms at about the age of 40. (2) Many adults with adeno-parenchymatous goitre give history of enlargement starting at puberty.

Treatment—Adoption of hygienic measures. Elimination of septic foci and administration of harmless iodine preparation such as syrup ferri iodide (1 dr. t. d. s.) is all that is needed. The use of thyroid extract and Lugol's solution is discouraged because a few examples of Graves' disease have been observed by its use.

ADENO-PARENCHYMATOUS OR ENDEMIC GOITRE—The principal regions in which endemic goitre occurs in this country are the districts bordering the foot hills of Himalayas round about Kangra, Hoshiarpur, Kalka, Murree, Dalhousie, Dehra Dun and Nainital in U.P. It sometimes occurs in non-mountanous regions also. For instance, the condition is not uncommon in Tahsil Kabirwala in Multan District and is also prevalent in some parts of Jhelum District.

The condition is in some way related to an iodine deficiency in food and water. The theories are:

(*a*) Kocher ascribes the origin to the presence of chemical substances specially the calcium carbonates in the water which are dependent upon a geological character of the soil through which the water percolates.

(*b*) Toxic infective theory of Mc Carrison—He assumes the existence of specific intestinal flora, the toxic products of which exert their mischief on the thyroid gland. He quotes cases in which intestinal antiseptics cured the condition.

(*c*) Theory of iodine dificiency which asserts that the condition is due to lack of requisite iodine in food and water. There is at present a tendency to combine the last two theories. It is suggested that although there may be an adequate amount of iodine in the food and water, the iodine is rendered unassimilable by the presence of specific intestinal flora or through lack of fat soluble vitamin A. Some goitre-producing factor, a glucoside, liberating cyanide *e.g.* cabbage has also been blamed.

A striking inverse relationship between contents of iodine in diet and proportion of goitrous individuals among the population has been observed. Japan is practically non-goitrous because iodine consumption of Japanese is more than double of any other nation. The close proximity to sea undoubtedly increases the intake of airborne iodine. It has been suggested that one must live within three miles of the sea to derive perceptible benefit from air borne iodine.

Morbid Anatomy—Hyperplastic changes in the gland are responsible for the formation of circumscribed nodules varying from microscopic size to enormous dimensions, leaving in between normal or atrophic parenchyma. The nodules contain, in early stages excessive amount of colloid which distends the acini, while the epithelium remains flat. This forms a colloid goitre. Through the absorption of the septa several acini may coalesce and form a large cyst—cystic colloid goitre. In some cases the hypertrophy involves the isthmus. In others outlying accessory nodule of thyroid tissue constitutes the bulk of the swelling and this may extend a considerable distance from the position of the normal thyroid reaching even behind the sternum into the thorax forming retrosternal goitre.

Clinical features—The deformity of the neck is a leading complaint. The swelling moves on deglutition and is smooth in early stages but later on may become hard and nodular.

Pressure on the trachea may develop. In unilateral enlargement the trachea is pushed to the opposite side and there may be dyspnoea. But in bilateral enlargement the trachea is markedly compressed and dyspnoea may be severe. In advanced cases trachea may be reduced to mere anteroposterior slitscabbard trachea.

Treatment—Preventive. In the goitrous regions provision of pure water and table salt containing iodine I in 100,000 has reduced the incidence of the disease. Even the cows and sheep which got goitre in the goitrous region responded to the prophylactic treatment. Kocher advises to boil all water supplies for drinking in the goitrous regions. The administration of thymol and other antiseptics also arrest the disease.

The iodine is excreted in milk and so exhibition of iodized salt to pregnant mothers prevents the onset of goitres in children.

Surgical treatment is advised where there is much deformity or symptoms of pressure on trachea are present. A good radiograph will show the degree of tracheal displacement.

The gland is exposed and a wedge resection performed.

THYROTOXIC GOITRE—Although many of the symptoms of thyrotoxic goitre may be produced by excessive doses of thyroid extract, the disease is probably due to an abnormal rather than excessive normal secretion.

PRIMARY GRAVES' DISEASE—It is not very common in this country. The symptoms appear in third or fourth decade of life but may occur earlier or later. Females are affected six times more than the males. The onset usually dates from a crisis in patient's life —a mental shock etc. The disease progresses by acute exacerbations and remission. It is well to remember that the exophthalmos or the goitre may be absent but seldom both. The thyroid gland is enlarged evenly, very vascular so that the "thyroid thrill" is usually obtained. The patient wastes and is subject to attacks of diarrhœa. Muscular weakness progresses as the disease advances and marked cardio-vascular and nervous symptoms appear. There is tachycardia. The blood pressure is raised and the slightest excitement accelerates the circulatory turmoil. Eventually auricular fibrillation sets in. The patient is restless and highly strung. The extended hands shake and the tongue is tremulous in some. Hysterical weeping is one of the early manifestations of mental abnormality. Metabolic rate is considerably raised. The skin is warm and moist.

Morbid Anatomy—There is proliferation of the epithelium of the alveoli which are small and closely packed together. There is no colloid in the acini. There may be intrapapillary growth or cushions projecting into the alveoli. The interalveolar connective and fibrous tissue is much more vascular. The whole picture is one of secretory activity and the colloid being passed into the blood stream as fast as it is formed and not stored up at all. There is diffuse hyperplasia of the entire lymphatic system notably the thymus. The reticulo-endothelial system is hyperactive. In severe cases there is leucopenia. In this condition there is production of abnormal thyroglobulin and the abnormality consists in deficiency of thyroxin and di-iodotyrosine radicals with a proportionately greater contents of thyroxine. The thyroxine contents is inversely proportional to basal metabolic rate. It would appear that conversion of tyrosine radical to those of di-iodotyroxine and especially of thyroxine is interfered with. The excessive demand on the part of the tissues for thyroxin accounts for proliferative changes in the gland cells and concommitant loss of colloid.

There is increased concentration of iodine in the blood and greater excretion in the urine. Excretion of calcium and creatine is elevated in the urine while cholesterol in blood is decreased. Liver is depleted of its glycogen and there is low sugar tolerance and often glycosuria. Prolonged decalcification may lead to calcoporosis.

Differential diagnosis is chiefly from simple goitre in patients with neurosis and from heart disease not related to thyroid.

Use can be made of iodine response in diagnosis. Clear cut improvement with fall in B.M.R. on iodine administration and with relapse on its omission, is almost pathognomonic of thyrotoxicosis.

X-ray, electrocardiography and estimation of blood cholesterol and blood creatine give useful information. There is increased sensitivity to adrenaline.

Diabetes mellitus is exaggerated by thyrotoxicosis. A latent diabetes may be made manifest by development of thyrotoxicosis and relief of thyrotoxicosis will ameliorate co-existing diabetes.

SECONDARY GRAVES' DISEASE—(*Toxic Adenoma or Plummer's Disease*)—This condition is seen in women over forty who have had a simple goitre or multiple adenomas for years. Although the symptoms are less severe than the primary Graves' disease the condition is progressive and remissions are absent. Cardiovascular symptoms are most in evidence and many of these patients have myocardial degeneration. Exophthalmos is always absent. These toxic adenomas occur at the lower poles and may even be retrosternal.

Treatment of Thyrotoxic goitre—Medical treatment should be tried for six months in the early cases. It usually consists in administration of Lugol's solution, digitalis, milk of thyroidectomized goats, bromides and rest in bed. Some authorities also advise x-ray treatment during this period. The results of surgical treatment are so good that when medical treatment fails or there is relapse after apparent improvement operation should be unhesitatingly advised.

The advent of thiouracil (which belongs to the sulphonamide group) bids fair to revolutionize the treatment of thyrotoxicosis. In a discussion at the Royal Society of Medicine last year Himsworth and others asserted that thiouracil is a potent weapon for overcoming thyrotoxicosis. Although the drug is not devoid of risk, an initial dose of 0·2 mgm. is recommended for the first 3-5 weeks and then a maintenance dose compatible with the well being of the patient is found. The drug is still on trial and the time will prove its ultimate utility and whether it can replace sound surgical measure. Himsworth and others have reported many cases where the benefit in their opinion appeared to equalize subtotal thyroidectomy.

Before operating the patient must be (1) fully iodized, (2) must be free from infections, throat and others, (3) must have good store of liver glycogen and body fluids, (4) must have had chest x-rayed etc., and (5) in cases of congestive heart failure the heart must have been well digitalized. Relative contra-indications—(1) Poor or absent iodine response, (2) rising B.M.R., (3) presence of severe complications chiefly cardiac, (4) extreme intoxication indicated by very high B.M.R. or severe nervous symptoms, (5) old age, (6) psychic manifestations, (7) marked malnutrition, (8) marked myasthenia or muscle atrophy.

Pre-operative treatment consists in:—

1. Absolute rest in bed and ensuring peace of mind. 2. Iodine in the form of Lugol's solution (5 per cent iodine in 10 per cent potassium iodide watery solution) 10 minims three times a day in milk or orange juice. The best results of iodine therapy are obtained between 8th and 12th day. It is characterised by gradual drop in pulse rate to a constant level, disappearance of nervousness and lowered B.M.R. and general improvement. 3. Lactovegetarian and a high calory and high carbohydrate diet and copious drinks of fluids. In improving nutrition vitamin B is important. 4. Prominol ½ grain or bromide 15 grains at bed time daily. 5. Digitalization by giving 30 minims four hourly. 6. If œdema is present 30 to 60 grains of ammonium chloride three times a day or novasurol intravenously. 7. If delirium—morphine in sufficient quantities to keep the patient quiet.

Operative treatment—For success, perfect team work between the physician, anæsthetist and surgeon is necessary.

Subtotal thyroidectomy remains the operation of choice for cases of diffuse toxic goitre, a strip of thyroid tissue being left on either side in front of the hilum of gland (the site of entrance of the inferior thyroid artery) as a protection to the recurrent nerve and the parathyroids. In nodular toxic goitre resection-enucleation of the adenomatous masses and of atrophied or hyperplastic gland tissue is the procedure of choice.

In cases where after a course of iodine treatment the B.M.R. does not come down, preliminary ligature operation is advisable.

Preliminary Ligature Operation—The inferior thyroid artery is the best vessel to ligate because it is the blood supply of the gland proper and is usually a larger vessel than the superior, which is distributed chiefly on the surface of the gland. The trunk of the artery is tied through an incision behind the lower part of the posterior border of the sternomastoid. Others prefer to ligate the superior artery at the upper pole of the gland, because of the more superficial and readily accessible position.

Crile believes in washing out the wound just prior to closure with .10 per cent saline solution, claiming that this removes free colloid and so reduces the amount of post-operative absorption and the tendency to subsequent reaction, and this also shows up any bleeding point which may be obscured by small pieces of blood clot.

Complications—In addition to cardiovascular complications of thyrotoxicosis, Dunhill discusses glycosuria, mental derangement, extreme emaciation, and skin complications, notably pruritus.

In severe hyperthyroidism there is a diminution in the glycogen reserve in the liver, and hepatic insufficiency is a factor in fatal cases. Strict hæmostasis is imperative.

Anæsthesia in the surgery of thyrotoxicosis—A combination of rectal paraldehyde or avertine with morphine, and local infiltration with novocaine or procaine hydrochloride 1 per cent are well recommended. The paraldehyde is given in saline 1½ hours before the operation is begun—5 drachms being a usual dose, but this is increased or diminished according to the build and condition of the patient. Inhalation anæsthesia is avoided. Others prefer only local infiltration with morphine in association with or without gas and oxygen.

Postoperative Complications—Hæmorrhage—may be very profuse. Urgent dyspnœa may occur as a result of blood clot pressing on the trachea. The wound should be opened and the bleeding vessel ligated. Acute hyperthyroidism is manifested by increased pulse rate, restlessness and vomiting. It is combated by intravenous saline and glucose to which 20 minims of Lugol's solution is added. Tetany occurs after 24 hours and is due to accidental parathyroidectomy. Calcium and parathormone are given. Huskiness of voice is due to traction or damage to the recurrent laryngeal nerve.

NEOPLASM—I. *Benign*—They are adenomas frequently solitary but two or three may be present. The junction of isthmus with one of the lateral lobes is the favourite site. If in the lower part they often become retrosternal.

Pathologically they are of two types:—1. Fœtal adenoma and 2. Adult adenoma.

This classification depends entirely upon the type of the cell per cent. In fœtal type the acini are packed with cells. There is no colloid. It occurs in young patients and does not undergo cystic degeneration. The adult type is like the ordinary adult thyroid tissue, contains colloid and frequently undergoes cystic degeneration. Both varieties are encapsulated.

Clinically there is a rounded swelling in the thyroid region. Dyspnœa may be present if the tumour presses on the trachea; it is first evident on exertion but later on troubles the patient at night. Sudden hæmorrhage in adenoma may cause urgent dyspnœa and threaten life (I had a case of this). It may also occur in large retrosternal goitres. Toxic symptoms may supervene. There may be alteration in voice due to pressure on the recurrent nerve. It is not necessarily the result of carcinomatous change.

Radiography is of great help in diagnosis. Firstly, if the adenoma is calcified and secondly in retrosternal goitres particularly in which the walls are often calcified. It will also show the tracheal deformity.

II. *Malignant*—It is of three varieties: 1. Carcinoma. 2. Papilliferous adenocarcinoma. 3. Scirrhus carcinoma.

Carcinoma of the thyroid usually commences in the pre-existing adenoma. Hence the cardinal rule is that every thyroid adenoma should be enucleated early. Some apparently benign tumors on microscopic examination turn out to be malignant, and *vice versa, i.e.,* metastasize. Whenever the carcinoma is suspected, early operation is the only hope. The following points will indicate its clinical nature:—1. Progressive increase in the size. 2. Fixity to the surrounding structures. 3. Uneven and nodular surface. 4. Hard consistency. 5. Husky voice due to involvement of the recurrent nerve. 6. Progressive pressure effect on trachea and finally its actual infiltration causing blood tinged sputum. 7. Involvement of lymph glands. 8. Metastases which are usually blood borne occur in the lungs and bones particularly the skull, sternum and mandible.

It is interesting to note that metastasis of the malignant thyroid usually function for the body's need after removal of the primary growth and hypothyroidism is practically unknown.

Treatment—Successful removal is possible only in early stages. Inoperable cases can only be treated by deep x-ray therapy. Radium has not proved of much use and tracheotomy as a palliative measure may be required to relieve dyspnœa. Sarcoma is very rare indeed. The clinical points are the same as in carcinoma.

INFLAMMATION OF THYROID is very common. Acute thyroiditis is usually secondary to tonsillitis or pneumonia. Suppuration is rare.

Tuberculosis and syphilitic affections are still rare. A special form of chronic inflammatory lesion called ligneous or woody thyroiditis is essentially a granuloma and is seldom seen here.

TOTAL THYROIDECTOMY

In patients without thyroid disease it is helpful sometimes for the sake of one or more of its several actions to give thyroid hormone; so too in certain others it may be desirable to withdraw it. The latter can be done by performing total thyroidectomy, which Blumgart, Levine and Berlin have shown is a feasible practical procedure.

Total thyroidectomy to be sure causes athyreosis and myxœdema. It brings about a state of affairs in which the organism lives at a slower pace in all departments of existence. If certain organs are weakened through disease and inadequate for the task demanded by normal living, they may become relatively adequate if the rate of living is reduced, and the burden on the said organs thus lessened.

Of course, myxœdema of itself is undesirable, but having totally ablated the thyroid one can then carry a patient at any metabolic level desired by giving a suitable ration of thyroid.

In congestive heart failure results to date seem to show that in well selected cases there is about an even chance of obtaining worth while benefit by total thyroidectomy. Selection of cases also is very far from easy because of the multiplicity of contra-indications.

In angina pectoris the prospect of benefit is considerably greater. However, even here there are probably very few patients who should be subjected to the procedure. For intractable angina the results by the alternative method of paravertebral alcohol injection compare very favourably with those by thyroidectomy. This procedure lacks the disadvantage of producing myxœdema. The nature of the very early relief of angina following total thyroidectomy has not been conclusively determined. There are some conflicting views on the subject. Whatever its cause, its occurrence is a clinical fact.

Total thyroidectomy for diabetes mellitus rests on sound theoretical consideration. In practice, in one case it seems to have been worth while in another it was not so.

One case of lymphatic leukemia has been treated by total thyroidectomy with symptomatic benefit.

APPLICATION OF SACRAL ANAESTHESIA IN GENERAL SURGERY

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Sacral anæsthesia or caudal anæsthesia as it is also called is an epidural anæsthesia, anæsthetising the region supplied by the sacral nerves.

Cathelin was the first to use the sacral anæsthesia not as a means of surgical analgesic measure but he used it in the treatment of pelvic and sexual neurosis by injecting analgesic solutions into the canal and also for analgesizing the pelvic outlet in delivery. He injected sterile water, sterile normal saline, novocaine and other medicated solutions.

The sac of the enveloping duramater terminates opposite the 3rd sacral vertebra and the sacral canal below this is occupied by connective tissue and the sacral nerve trunks. It is the nerves within this area below the outside of the dural sac, which are acted upon by the solutions injected within the canal.

The analgesic solution injected does not enter the subarachnoid space but bathes the nerves on their way to the intervertebral foramina surrounded by their dura and causes interruption or block.

The sacral anæsthesia, it can be seen therefore is an epidural anæsthesia and not intraspinal and therefore, is quite safe without the danger and disadvantages of the spinal anæsthesia.

During recent years this method of obtaining anæsthesia of the nerves arising from the sacral canal has become so firmly established that it is the method of choice in the surgery of the perineum. Any operation on the perinoum can be undertaken with great ease without any risk whatsoever. The following operations for instance have been performed quite successfully:

Fissures, piles, fistula in ano, rectal polypi, prolapse of the rectum, internal and external urethrotomies perineal pithotomies, hydroceles, orchidectomies, amputations of the penis, perineal prostatectomy, vaginoplastics, vesicovaginal fistula, rectovaginal fistula, vaginal lithotomy, trachelorrhaphy, dilatations and curetting, amputations of the cervix, pelvic abscesses, suprapubic cystotomies and prostatectomy in combination with local infiltration of the abdominal walls, cystoscopies and application of low forceps.

Especially it is the ideal method in the surgical treatment of fistula in ano and hæmorrhoidectomy. In these operations it has the decided advantage of relaxing the anal sphincter to such an extent as to render dilatation easy and complete without danger to its muscular structures. It is the usual practice with me to tell patients after the anæsthesia is established that I am only examining them, they will feel the pressure of fingers only and that just before the operation they will be given a few whiffs of chloroform. Under this impression the actual operation finishes off without the least knowledge on the part of the patient and many a patient has asked me as to when the operation will begin and when he is told that the operation is finished, he was surprised and could not believe it. So, you can as it were, steal the piles from him without his knowledge.

Sacral anæsthesia finds its real test in the posterior resection of the rectum for which it has now been adopted by many surgeons. When correctly done it is of great advantage in as much as it lessens the risk of operations.

At the sacrococcygeal junction, there is a depression which is known as the sacral hiatus. The patient is made to lie prone on the abdomen. The sacral hiatus or the depression is felt in the midline by the tip of the finger. After thoroughly cleansing the area and sterilising it in the usual manner it may be anæsthetised by novocaine or under ethyl chloride spray. A good sized long needle is introduced through the hiatus upto ½" to 2 inches in length. After the needle properly enters the canal, 1 per cent novocaine from 40 to 120 c.c. can be injected. I usually inject from 40 to 60 c.c. If the needle is in the proper position the fluid from the syringe is easily injected without any resistence and in 3 to 4 minutes the patient complains of numbness in the inner side of both thighs. This is an indication that the anæsthetic is acting. It takes full 20 minutes for complete anæsthesia to set in. If the operation is started early the patient may resist and not co-operate. So, full 20 minutes should be allowed before the operation is attempted. The anæsthesia usually lasts from 30 to 45 minutes. If anæsthesia of longer duration is required, more quantity of the solution should be injected, which should be approximately determined before the injection.

In a certain number of cases the sacral hiatus cannot be punctured in the midline owing to the presence of a spur. It may be very difficult to locate it in very fat individuals. Occasionally angulation of the sacrum is such that the needle fails to enter more than 2 or 3 cm. which is not sufficient to produce anæsthesia of certain regions particularly the base of the bladder and its neck. In other cases abnormalities of the sacrum in shape and contour render the puncture of the sacral canal hazardous. Infection of the region prohibit the use of the caudal block because of the danger of infecting the canal. Sometimes the nerve sheathes may be so thick as to reduce by absorption the strength of the solution before the nerve fibres are reached. Hence in view of the above unfavourable factors, the anæthesia may fail in 5-10 per cent cases to which may be added the failure to establish it for lack of skill.

Regarding pre-medication there is nothing special except that morphine and atropine are routinely given ½ hour before the operation.

If the patient is extremely nervous or high strung he may not co-operate with the surgeon at all and interpret the pressure sensation of fingers as pain and thus resist and make the operation impossible. Such a patient is psychically a bad case for any local anæsthesia and then there is no other go but resort to general anæsthesia or some other method.

Another disadvantage is the danger of the sacral canal getting infected but I have never met one so far.

(Continued at foot of page 328)

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SNAKE-BITE

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(Continued from previous issue)

Mechanism of Snake-Bite

In the resting position, the fangs of a snake are lying in the mouth pointing backwards and slightly upwards. While holding vipers by the head, the worker has to be careful not to press too hard the floor of the mouth, otherwise the fangs may pass through this floor and enter the skin of the worker thus completing the action of snake-bite. During the act of biting, due to a complicated muscular action, the fangs are rotated forwards, the entrance hole, situated at the anterior surface of the upper end of the fang fits itself to the end of the venom duct. The muscle, covering the venom-gland presses the gland like a vice and venom flows down the duct and fang into the tissues of the victim. The whole act is accomplished in a fraction of a second. It is so instantaneous in the case of a viper, that an onlooker may fail to notice it. Acton and Knowles put a daboia and a rat in a cage to see how the snake bites. In a minute the rat went into fatal convulsions because the viper had bitten it without being seen doing so.

The bite of the colubers (cobras and kraits) is not so instantaneous. The coluber may hang on for some time, probably to insure that the victim gets a surely lethal dose. There are however some cases on record in which the viper also had to be snatched away from the bitten area with some difficulty.

What is a Good Bite and the Dose Given with it

As mentioned above, if the central hole of the fang and the end of the venom-duct do not fit well into one another, the venom instead of flowing into the fang, and from it into the wound, will trickle into the mouth of the snake. The case will be one of snake-bite but not one of ophitoxæmia. That is one reason why a medical attendant should find out if symptoms of venom introduction are present, before he decides upon any method of treatment. The victim generally tries to get away from the bite. The mouth of the snake cannot grasp a whole limb. It often bites in an oblique manner, and may be able to thrust only one fang. The presence of the marks of the fangs cannot be a guide for the identity of a snake. The cobra may not leave any mark at all. Krait's fangs are still finer. The cobra injects 10 to 15 lethal doses in one bite, the daboia and krait 2 or 3. Therefore, entrance of only one fang into the tissues is enough to give a good bite.

The lethal dose of the dry venom of the cobra is 15 mg. or one-fourth of a grain or one-eighth of a *ratti*. It injects during one good bite 150 to 225 mg. or 10 to 15 lethal doses. Cobra believes in taking no risk.

Action of Snake Venoms

Snake venoms consist of proteoses, albumins, globulins, enzymes, and water. The colubrin venom is a golden yellow thick syrupy liquid, which solidifies on drying into similarly coloured shining crystals. The viperine venom is an opalescent watery solution, of a very thin consistency.

The dried venom if kept away from moisture, say on strong sulphuric acid or on calcium chloride or in sealed tubes remains potent indefinitely. If it absorbs moisture, it loses its potency. Snake venom is bitter in taste and if rubbed on healthy skin, one gets a soapy feel. It is not absorbed from the intact skin and intact mucous membrane. It is readily absorbed from the abraded skin, abraded mucous membrane, and subcutaneous tissue. Therefore there is no harm in its falling on intact skin and in sucking it, if the mucous membrane of the mouth is healthy.

It is destroyed by the digestive juice of the stomach. I put a lethal dose into the stomach of a dog, no symptoms developed while a similar quantity when introduced below the skin, invariably killed the animal.

On boiling, some constituents of the venom coagulate like the white of an egg. I boiled the solution of the lethal dose for a dog and injected the uncoagulated portion into the dog. The dog did not die, but the area of injection showed signs of irritation in the form of redness and swelling which disappeared in a few days' time.

Snake venoms are divided into 2 groups, colubrine and viperine. Colubrine venoms include venoms of king cobra, the common Indian cobra, the common krait, the banded krait, and the coral snake. Viperine venoms include venoms of the pitless vipers (the Russell's viper and the echis) and the crotaline vipers or the pit-vipers.

In general snake venoms kill the victim by paralysing either the central nervous system or the vascular system in other words each venom consists of two components, the neurotoxic, and the hæmovascular paralysant. In the colubrine group, the neurotoxic component predominates which depress the whole central nervous system, produces paralysis of lids, lips, cheeks, throat, larynx and kills by paralysing the respiratory centre. The hæmovascular component is smaller in amount in the colubrine group of venoms. It is a hæmolysin, which is responsible for oozing of hæmolysed blood from the bite.

In the viperine group of venoms the hæmovascular component predominates, while the neurotoxic component is absent or present in a very small quantity. It consists of a fibrin ferment, an antifibrin ferment, a hæmolysin and a hæmorrhagin. The fibrin ferment produces death by coagulation of blood and embolism, if the fang happens to enter a vein. This is the component, used as a hemostatic in therapeutics. The antifibrin ferment reduces the coagulation-power of the blood. The hæmolysin destroys the red blood cells. These 2 latter are responsible for oozing of hæmolysed blood. The hæmorrhagin destroys the cells of the capillaries and is responsible for the internal hæmorrhages, hæmorrhage from different orifices of the body and hæmorrhage under the skin.

All the components of the viperine venom are highly irritant to the tissues, and produce irritation, swelling, discoloration, necrosis and pain.

The viperine venom kills by lowering blood pressure due to its hæmorrhagin, producing exhaustion from continuous hæmorrhage, and in prolonged cases by superadded sepsis or tetanus.

Diagnosis of a Case of Snake-bite

Determine whether the victim has been at all bitten and if bitten, by a snake or any other animal like the scorpion. If the snake that has bitten is available, see it and decide its entity. If it is non-poisonous, no specific treatment is needed. Wash the wound with clean boiled water, apply a clean bandage, and assure the patient of the harmlessness of the wound. This will counteract shock and avoid death.

If the offending snake is found to be poisonous, determine if the venom has been injected into the victim or not.

The word PODS sums the symptoms of a venomised wound. P stands for pain, O for oozing, D for discolouration and S for swelling.

In a venomised wound pain is out of proportion to the injury done by the bite. It is very acute and starts at once. In a non-poisonous bite, the pain is as much as that produced by a prick from a thorn or any fine object.

Every snake venom, specially that of the vipers reduces the coagulation power of the blood, hence after a poisonous bite, oozing of a bloody fluid may continue for some time. Continued oozing of blood if present is a sure sign of the presence of the venom.

Venom causes death of the tissues, extravasation of blood occurs and causes discolouration, which if present is a sure proof of the presence of the venom.

Swelling starts immediately after a poisonous bite. In a non-poisonous bite swelling is practically absent.

The general idea that a snake-bitten person sleeps, is true only in the sense, that the venoms of the cobra and the krait depress the brain, and the victim gets paralysis of tongue, lips, speech and last of all of swallowing. He never sleeps. He is conscious till about the end. Death from the cobra bite is a very painful death. Most of the deaths in snake-bite are due to the cobra.

Symptoms and Signs of Colubrine Poisoning

Local signs of the introduction of venom into the tissues start immediately after the bite. Oozing of hæmolyzed blood from the wound is present in less than half the cases, and when present, is a sure sign of the venom having been introduced. The antifibrin ferment prevents coagulation of blood and sealing of the punctures. Hæmolysin dissolves the envelops of the red blood cells and causes hæmolysis. This oozing may go on for hours, and is more frequent in viperine than in colubrine poisoning.

The neurotoxic component of the colubrine venoms produces weakness of the legs, paralyses the bulbar nuclei causing paralysis of lips, tongue, cheeks, throat and larynx, and kills by paralysing the respiratory centre. It is a general depressant of the central nervous system.

A few minutes after the bite, the victim feels weak in his legs, he staggers and sits down. After some time he is unable to sit, and lies down. The lips fall away from the teeth, swallowing becomes difficult, and saliva collects in the mouth. Snake bitten subjects have been seen trying to remove the saliva from their mouth with their fingers. Due to failure of the lips, the labial letters cannot be pronounced. A man named Baon Das pronounced his name as Aon Das.

The eye-lids droop. To see people, the victim has to turn his head back. His neck cannot hold the weight of his head and consequently droops. Respirations go on decreasing in number until they finally cease. The heart goes on beating for some time after the respiration has ceased.

The bitten area is swollen, and often discoloured. It is not always possible to see the fang-punctures. If an incision is made, the subcutaneous tissues are found to be infiltrated with hæmolysed blood, having assumed a jelly-like appearance.

Last year at the instance of my friend, Syed Abdul Rahman, Professor of Physiology of our College, I injected into the lower third of the medial aspect of the left thigh of a dog, one lethal dose of cobra venom to see the local changes produced by the venom. On post-mortem we say that the above mentioned infiltration had taken place around the whole thigh, and under the skin of the lower three fourths of the left half of the abdomen. The lethal dose injected into this animal was only 5 milligrams, while an adult cobra injects in a good bite 150 to 225 milligrams.

The action of cobra venom (which is a good representative of all colubrine venoms) can be easily remembered from the word cobra. CO stands for cord, BRA for brain. Cobra venom depresses brain and cord. Again COBR stands for coagulation of blood reduced and A stands for asphyxia, the mode of death.

Symptoms and Signs of Viperine Poisoning

The vipers are divided into pitless and pit vipers. In India venom of the pitless vipers is fatal but that of pit-vipers is toxic but not fatal. In America, the venom of the pit-vipers is fatal. In India we are mostly concerned with the venom of Russell's viper and that of the echis, because that of the Indian pit-vipers does not cause fatalities. It may cause swelling, fever and some necrosis.

The viperine venom is known to contain two components—a neurotoxin which is small in amount and a hæmovascular paralysant which predominates. Viperine venom is much more irritant in character than the colubrine venom. It produces more pain, more burning, more swelling and more oozing. There is a fall of blood pressure due to paralysis of capillaries and depression of the vasomotor centre. Due to the irritant nature of the venom, there occurs a small white cell infiltration around the deposit of the venom, and a further absorption of the venom is lessened. In other words a protective wall is formed around the deposit of the venom and the local effects of the venom tend to persist for weeks and months.

A man aged 45 came to me from Dharur near Viqarabad. His history was this. Two months before, one evening after sunset, he went into a field to get some hay. He was not wearing shoes, he felt as if he

put his foot on a soft thing like cattle-dung. He heard a hissing sound and was bitten on the middle of the back of his right foot. The animal's body touched his leg and it appeared to be as thick as the wrist of a 10 year old boy. He knew it was a snake. He returned home. In about 12 hours the leg swelled up to above the knee. There was a burning pain in it. He did not remember if there was any oozing. He got no paralysis. He applied some medicine. In about 10 days time most of the swelling was gone, but some swelling persisted. Being a labourer he has to walk. When he goes about a mile, his foot swells, there is a burning pain, and heaviness of the foot all this compel him to remain at home.

On examination I found that there was a scar one-quarter inch square at the bite, and there was œdema of the foot extending about two inches above the medial malleolus.

The absence of the history of paralysis ruled out the colubrines. Of the vipers, the pit-vipers are not met with in our Dominions. Out of the pitless only two are found, the Russell's and the echis. The latter is never thicker than one finger. The offending snake left behind is Russell's viper.

Considering the above mentioned character of this venom, I assumed that some venom was still present at the bitten site and was responsible for the local condition persisting.

One tube of 10 c.c. concentrated Kasauli anti-venomous serum was given intravenously, diluted with 10 c.c. of distilled water. The patient was advised to come after 3 days.

He returned to report, that most of the œdema was gone, he could walk more than two miles without feeling any burning or heaviness. For the remaining œdema I did not think it necessary to inject serum, instead I gave him, two intravenous injections of calcium gluconate at an interval of two days to counter-act the damage done to capillary walls. The whole œdema disappeared.

This case was very instructive. Knowledge of the external characters of the snakes, and of the pharmacological actions of different venoms helped in settling the identity of the snake and with it the treatment.

Cases are on record in which gangrene of the soft parts has occurred, leaving the bones bare, due to local destructive action of the viperine venom.

The vipers have got a habit of jumping and biting. Wounds inflicted by them may be higher on legs. The colubrides (cobras and kraits) generally direct their strokes downwards and wounds inflicted by these are generally found on the feet and lower parts of legs.

The vipers may bite even without a provocation. It has been seen that the living species of echis kept in glass-walled cages in the Museum of the Bombay Natural Society in an attempt to strike at the visitors strike against the glass-wall of the cages, and the drops of the venom are seen trickling down along the inner surface of the glass-wall.

The Russell's viper generally does not try to get out of the way of a man who comes towards him, while a cobra will generally avoid and get out of the way. The krait is a very dull and apathetic animal, very much averse to biting. It bites when seriously troubled or pressed. Mr. Prater reports that some boys made a noose of a rope, put it around the neck of a krait, and brought it home as a trophy. The snake never minded the playful treatment, and took it in a sporting spirit.

Differentiation between Symptoms of Snake-bite and Symptoms of Scorpion-bite

In some cases, when the offending animal is not seen, there is some difficulty in settling, whether the offender is a snake or a scorpion. I have seen a case of scorpion-bite, where the victim was a young educated man. He came to me at 5 A.M. on a cold morning. He was pale and drenched in cold clammy perspiration. There was no sign of paralysis. Colubrides were therefore ruled out. Of the local signs of snake bite only œdema was present, the rest *viz.*, oozing and discolouration were absent. These two are more prominent in a viper-bite, for which it could be mistaken. The absence of either of these two ruled out a viper-bite as well. If any of the definite symptoms of snake poisoning such as bleeding, discolouration, or paralysis are seen, no difficulty arises.

Differentiation between Symptoms of Snake-bite and Symptoms of Shock due to Fright

That fright of death can cause fatal shock, has been proved. It is more easily produced in timid, superstitious and ignorant natives of our country. Persons have died after the bite of a 'bis copra' (Varanus bengalensis) one of the monitor lizards, and absolutely harmless. Death has occurred after the bite of brahminy lizard, which is a perfectly harmless creature. People have died after the bite of a house snake (Lycodon aulicus).

As mentioned before the snake-venom either attacks the central nervous system or the cardio-vascular system. A victim of ophitoxæmia remains conscious up to a very late stage of toxaemia. In cases in which the fang happens to enter a vein and deposits venom directly into the circulating blood death is instantaneous. Persons who get unconscious, immediately after being bitten by a snake, do so only through psychological shock due to fright. The least time that a lethal dose of a venom takes to be absorbed and be able to exert its toxic influence is 15 to 20 minutes. Besides the local symptoms produced, the colubrine venom will produce gradual weakness ending in paralysis, the viperine will take even longer to produce its effect on circulation, but neither will produce instantaneous shock and fainting.

Shock due to fright can never be confused with symptoms of the viperine toxæmia, as the local symptoms of a viper bite are very prominent. The victim may die instantaneously due to intravenous deposition of the viperine venom, but there is no possibility of instantaneous shock and fainting due to viperine venom. If a person faints and is shocked instantaneously after a bite, it is definitely a psychological condition of shock due to fright. The unconsciousness of psychological shock may be confused with

the depression and paralysis produced by colubrine venoms.

Symptoms met with in fright and colubrine poisoning require emphasizing especially as they are in almost every detail strikingly different, and as a result require completely different methods of treatment.

Fear operating through the nervous system mainly affects the heart. The symptoms may vary from a transient pallor and giddiness to syncope of so profound a nature that unless combative measures are speedily employed the condition may pass insensibly on to death.

Cobra being the type of a colubrine snake, the toxæmia produced by its bite exerts its main force upon the nervous system, and principally depresses the respiratory centre, the heart remaining unaffected. The constitutional effects seen in the two cases are as follows:—

Fright	*Cobra poisoning*
1. Onset of weakness often sudden.	Very gradual
2. Involuntary prostration, often the patient falls in a faint, and is brought in this state to hospital.	Recumbency voluntary after some time owing to gradual loss of power of legs. The victim often walks to hospital by himself or with help.
3. Complete or semi-unconsciousness.	Consciousness not impaired.
4. Syncope	Heart not affected
(*a*) Pallid face	(*a*) Face natural at first, livid later on.
(*b*) Cold clammy skin	(*b*) Skin warm.
(*c*) Feeble or imperceptible pulse.	(*c*) Pulse of normal force or regularity.
5. Breathing shallow, sighing, and weak and unduly frequent (30 a minute or more).	Gradually becomes more and more laboured and quickened, gasping towards close.
6. No paralysis.	Paralysis, gradual weakness of legs mounting upwards to trunk and head. The head droops. The eyelids droop. Swallowing becomes difficult, the lower lip falls away from the teeth and saliva dribbles from the mouth. Articulation also becomes difficult.
7. Death from cardiac depression.	Death from respiratory paralysis.

The symptoms of fright often very speedily declare themselves, and far more speedily than is ever the case in snake poisoning. In some reported cases the patient is seen or is brought to hospital in a senseless or nearly senseless condition, it may be a few minutes after the bite. Often this unconsciousness is taken as a result of absorption of venom, and remedial measures are taken accordingly.

The history that a person got senseless immediately after the bite of a snake, is proof sufficient for the deduction that the condition is due to fright.

Treatment

It is estimated that 50 per cent of deaths are due to shock. If every one is taught distinction of snakes, these 50 per cent deaths can be prevented. The remaining 50 per cent require a medicine, all the so-called snake-bite cures have been tried and found useless. The only potent drug is the antivenomous serum.

Treatment Advised by Director-General Research Institute, Kasauli

Instructions for the use of Concentrated Anti-venom serum (Concentrated Antivenene).

I. The Antivenene is a concentrate from the serum of horses immunized against the venoms of cobra and daboia (Russell's viper). One bottle of 10 c.c. is equivalent to 40 c.c. of the unconcentrated serum.

II. In the case of cobra or daboia bite the serum should be injected intravenously or subcutaneously. *It is four times as effective when given intravenously.*

III. Treatment of cobra bite.—(*a*) In the case of bites on the limbs apply a tourniquet in order to prevent and control the flow of poisoned blood and lymph from the area of the bite. In the case of the leg apply the tourniquet above the knee and in the case of the arm above the elbow. Do not keep tourniquet on longer than 30 minutes at one time.

(*b*) Make an incision at least ¼th inch deep over the fang wounds about one or two inches long and encourage bleeding to wash out the poison. Pour into the wounds a strong solution of permanganate of potash, apply a wet antiseptic dressing.

(*c*) Inject intravenously one or two bottles of 10 c.c. concentrated serum, or two or more subcutaneously.

When the biting snake is a large one or the symptoms continue the dose may be repeated.

If the case is seen late and when symptoms have developed intravenous injection should always be employed.

(*d*) Additional methods of treatment which may be used are the administration of coffee, and the injection of 1 c.c. of 1 in 1000 solution of adrenalin or 0·6 c.c. to 1·0 c.c. of pituitrin.

IV. Treatment of daboia (Russell's viper) bite —(*a*) The same procedure of the application of a tourniquet and incision and local treatment should be followed.

(*b*) The serum is more potent against daboia venom than against cobra venom. One bottle intravenously or two bottles subcutaneously will be sufficient unless a large dose of the venom has been injected, provided the serum is given early. If seen later double the quantity may be given.

As the venom is liable to be partly fixed locally at the site of the bite one quarter of the dose should be injected into the area where the fang wounds are seen.

(*c*) The injection of adrenalin (1 c.c. of 1 in 1,000 solution), ephedrine, one ¾ grain tablet in 1 c.c. of water, or pituitrin ½ to 1 c.c. hypodermically is of special value in preventing failure of the circulation in the case of daboia bite.

V. Treatment of echis bite.—As this antivenene neutralizes the hæmorrhagin of echis venom it should be used in cases of bites by E. Carinta (the phoorsa or saw sealed viper).

VI. Injection of serum.—The serum is sterile and preserved with an antiseptic. The syringe and needle used for injection should be sterilized before use. As it is essential that the injection should be given at the

earliest possible moment it will be sufficient for practical purposes to wash out the syringe several times with boiled water. Subcutaneous injection should be given under the loose skin of the abdomen or thigh. Intravenous injection should only be given by a medical man. If the serum is turbid the bottle should be placed in warm water at the temperature of the body for five minutes before opening for injection. The concentrated serum should be injected very slowly.

VII. The serum should be stored in a cool dark place and not exposed to light. The date of expiry is marked on each bottle and when approaching the expiry date the dose should be increased.

VIII. Persons who make use of the serum should communicate the results to:—The Director, Central Research Institute, Kasauli.

Testing the Efficacy of a Snake-bite Cure

Like all other poisons the snake venom must enter the animal body in a definite quantity in order to be fatal. This is called the minimal lethal dose. (M.L.D.). The M.L.D. for a man of about 65 seers is one-eighth of a *ratti* of the dried venom of the cobra.

The M.L.D. for a man of about 65 seers is one-eighth of a *ratti* of the dried venom of the cobra.

Different samples of venom differ in strength.

The fang of the snake is hollow. Its root comes below the mouth of the venom duct during the act of biting sometimes this falls and the venom falls into the mouth of the snake instead of entering the fang and through it the body of the victim. Therefore, biting experiments, conducted to prove the potency of cures are fallacious.

The standard test is to inject the M.L.D. of the dried venom into an experimental animal and then to use the drug to be tested.

No drug in the world has stood this test upto this time except the antivenomous serum.

Suggestions for Warding off Snakes

1. Remove unnecessary heaps of rubbish, bricks or stones. 2. Keep the verandahs of the house free from plants. 3. Dogs and cats by attacking the reptile and making a noise inform us of the reptile. 4. No body should sleep on the ground. 5. Avoid going into grass with bare feet, specially at night. When going out in the dark, take a lantern and a thick stick. The villagers' habit of thumping on the ground with a thick stick while walking at night is useful. The snake catches the thump through its body and bones and gets aside. 6. When going into a jungle, the use of leather leggings is good. 7. But gauze in the drains of your houses, so that snakes may not crawl into the house through these.

Value of so-called Snake-bite Cures

The snake species is said to have been in company of the human species, since Adam left paradise. Historical and philological records show that the word serpent is met with in the oldest literature of every country. Its capacity to cause instantaneous death has been the cause of the human's desire to get an antidote for its bite since antiquity.

The demand for a genuine and efficient cure is very great. The innumerable querries put by the public to the scientists are a definite proof of the interest that the public takes in the problem. The bite of a snake is a biological accident. Like all other biological phenomena, there is a lot of variation in every step of the process. The different steps are the will of the snake to strike, the will to strike with an open mouth or a closed mouth, the adaptation of the mouth of the venom duct to the basal hole of the fang, and the mood of the snake to inject venom.

The missing of any one of these different steps will make the bite non-poisonous and hence non-fatal. There are other factors which also interfere. If the snake has already bitten some animal and spent most of the venom of its glands, the dose may become sublethal. If the victim is wearing thick clothes or garters or boots, the fangs may not enter the skin. About 20 per cent victims of genuine cobra bite escape death without any treatment and about 30 per cent in daboia (Russell's viper) bite. Of all the bites taken together 80 per cent are due to non-poisonous snakes. Most of the claims made about curing snake-bitten subjects can be explained by these biological variations. Keeping all these scientific observations in view, a standard has been fixed, which a drug must reach before it can be pronounced as a true snake-bite-cure.

Snake venom retains its potency indefinitely, if kept in a dried form, in sealed tubes, or in a desiccator, on strong sulphuric acid or on calcium chloride, so that it may not absorb moisture and lose its potency. Venom is carefully collected, dried and kept thus. Lethal dose of each venom per kilogram of the body weight for different animals has been fixed. When it is desired to test the efficacy of a snake-bite cure, a surely lethal dose of venom is calculated according to the body-weight of the experimental animal, which is generally the dog, dissolved in water and injected intramuscularly. The specific is then administered according to the directions of the maker and the animal is watched. So far no snake-bite remedy, has stood this crucial test. Snake-bite serum is the only reliable remedy. The method described above eliminates all flaws which attend biting experiments.

What we need is that we should have at our disposal a remedy or remedies that may neutralize the effects of each and every sort of venom met with in the country. Only a fraction of the problem is solved. The present serum, as far as India is concerned, is only effective against the cobra and the daboia, and partly against the afai. There is no serum so far against the venom of the two kraits, the king cobra and the sea-snake which are fatal varieties. The venoms of the pit-vipers, although not fatal, cause a lot of disability such as pain, fever and swelling. The afai is very common in the Ratnagiri District of the Bombay Presidency, Rajputana and other sandy areas. Remedies against these varieties are also needed.

Further research must go on, to solve the remaining portion of the problem. Petty ideas should not be allowed to stand in the way of scientific progress.

We need a remedy which the layman may administer without difficulty. It should be cheap and

easily available. No case of snake-bite should be allowed to die.

Therapeutic Use of Snake-Venoms

All important life-saving drugs are potent poisons. Harm and benefit are relative terms. It is the dose of a drug that determines its usefulness or otherwise. To effect a particular change in the function or structure of a cell a drug must be present in the body in a particular concentration. The neurotoxin of the cobra venom stimulates the function of the respiratory centre in a minute dose, depresses it in a moderate dose, and paralyses it in a toxic dose. The first of these 3 actions (stimulation) has been shown by my own experiments (not yet reported). The depressive action of the cobra venom is utilized. The neurotoxin is separated and made free of other constituents *viz.*, hæmolysin, hæmorrhagin and enzymes. It is injected in small doses into people suffering from heightened irritability of their nerve-cells, particularly those concerned with perception of pain-sensation. Benefit is thus obtained in cases of neuritis, arthritis, chronic painful conditions, pains due to cancer and parkinsonism.

Neurotoxin is measured in mouse-units. A mouse-unit is that quantity of the venom which will kill a rat of 22 grams. in 18 hours after an intraperitoneal injection. In therapeutics 5 mouse-units is the average dose. The venom is supplied in dry ampoules, because it loses its potency if kept in solution. Solution is made in distilled sterile water at the time of injection. Injection is made deep into gluteus muscle. It is never given intravenously. Some people are sensitive and develop local irritation and pain. To avoid this it is advisable to start with 2-5 units. The therapeutic dose is three-thousandth of the human lethal dose, and does no harm. The preparation can be had from Messrs. Drug House India Ltd., 52 Shambhunath Pandit Street, Calcutta.

Because of its coagulant factor, the Russell's venom is similarly used to control hæmorrhagic conditions like hæmophilia and purpura. Several preparations are in the market. The above mentioned firm also supplies it.

Measure to Reduce Deaths due to Snake-bite

1. Make collections of specimens of snakes in every town. These should be multiple and exhibited at all important public places as mentioned above. 2. Educate public to distinguish between poisonous and nonpoisonous snakes. Remove all wrong ideas and superstitions from the mind of the people by means of public health teaching. 4. Provide snake-bite-serum in abundance to the public. 5. Include snake-bite literature in all schools and training courses. 6. Encourage research in problems not yet solved.

References

1. Gharpurey—The Snakes of India.,
2. Fayrer, J.—Thanatophidia of India.
3. Prater—The Snakes of Bombay Island and Salsette.
4. Wall—The Poisonous Terrestrial Snakes of India etc., 1917.
5. Wall—A Popular Treatise on the Common Snakes of India, *Journal of Bom. Nat., Hist. Soc.*, 1905-1919.
6. All articles published on the subject in the *Indian J. M. Research*, 1913-1944.

SPECIAL ARTICLE

MEDICAL MEN AND SCIENTISTS*

Present day scientists in the eye of a medical man

BIDHUBHUSAN BHATTACHARYA, B.SC., M.D.

To-day, when the War is said to be over, if we look to the world at large and compare with our mother land, India, we can see clearly our position as medical men. Although India was not a battle field, the casualty list will not probably fall short of any major combatant country and all this is due to famine and disease—both in most cases preventible. Malaria, cholera, dysentery and typhoid fever have taken the greatest toll and tuberculosis has been their common co-partner. But what is the real cause behind these diseases? Can't they be prevented? If we look to the free nations, what do we see from the medical stand point? I believe, although I am open to correction, that one day's war expense could have saved our millions. Bengal villages could have been regenerated and freed from these terrible devitalising and emasculating diseases only by a careful handling and expenditure —almost insignificant in comparison to the six years' war expense. I would like to remind you, just for an instance, about a negligible fraction of what can be done. Most of you, I am sure, have heard of Panagar-Mankar Anglo-Saxon colony, which has replaced about 30 poor villages and has been turned into a heaven, compared with the still remnant surrounding dying villages—and this has been possible in such a short time for the purpose of war.

Our leaders and social workers shout aloud—"back to villages"—but where are those villages of old—where to go—all villages to-day are infested with terrible diseases with no opportunity for culture, no food and none of the present day facilities of city life. Moreover, in the present state of our villages, will it suffice, for the medical men to flock together and instruct villagers to swallow quinine or mepacrine or some such drug morning and evening? I do not think such a method will do any good either to the people or the villages. Are these drugs the only necessity? Certainly not, the necessity is prevention of these diseases. If you go round the villages of Bengal, you will not only see human skeletons moving about, but the cattle, the lower animals and even the trees and plants all are suffering from diseases and malnutrition and moving towards anihilation.

The present day education and civilisation have given us all the artificiality in life, without health, without food and clothings, has taught us to earn our livelihood, if possible, by serving and pleasing our masters only and has necessarily forced us to live in towns. How can this be changed? To my mind this can only change if small towns are formed with all the facilities of modern industrialisation and education and if several villages are re-organised round each such town so that a completely new arrangement of villages are made. May be, there are other schemes, I am not aware of, which can give back the life, health and vigor, our ancestors enjoyed in villages.

*Lecture delivered at Bengal Provincial Medical Conference, Asansol, Nov., 1945.

It is rather an irony of fate, that we, medical men, who are and should remain mainly concerned with the amelioration of human suffering, are engaged to treat war casualties and efficient and able medical men are needed for conducting of such brutal warfare. The war in its train inevitably brings forth various epidemic diseases and famine. The scientists would do better humanitarian work if they would search for the cause of the after-effects of war, whether the epidemics are the result of the different chemicals used in war and consequent physico-chemical change in atmosphere or the lowered vitality of civilians by the nerve strain and food of less value, and if so, whether they can combat the evil effects, with effective anti-chemicals.

We, the medical men, are supposed to belong to the group of scientists—science has taught us whatever little show we present to the suffering humanity, our work is mainly consolation of the diseased and cure or relief where possible. But the best scientists of the world to-day, particularly chemists and physicists are engaged in researches for the discovery of dangerous and destructive implements of war, *e.g.*, atom bombs and death-rays—which will be able to destroy anything and everything throughout the length and breadth of any country or a continent wherefrom any opposition to their country may come. This opposition may not necessarily be military, may be social, agricultural, economic or any other trifling matter and lo! the resultant destruction explained in the name of teaching civilisation to the weaker and peace-loving people or the vanquished nations.

To my little brain, it is inconceivable that these scientists do not think of researches which would benefit the humanity of the Earth and will not at the same time cause destruction. But are not these scientists capable of utilising their energy in other ways which would rather bring happiness and peace to the world, in finding out measures to ameliorate human suffering, to prevent premature death and to eradicate the preventible diseases instead of searching after deadlier weapons? These scientists have turned into mere toys in the hands of their governmental bodies and possibly they have forgoten for the time being that other workers in the field may discover newer energy and the recent discoveries will be turned into dead-things—as has happened recently—the prize from the dynamite maker passes to the atom bomb discoverer. However, only time can show, as it flows through the *Mahakal*—which way the world is moving. We, in our utter helplessness—can only remind these powerful scientists and the world powers of to-day—in imitating our immortal Rabindranath—that when "the wheels of fate will turn round, what world will they leave behind".

I think these scientists, whose work is really wonderful, could have been much more serviceable and infinitely more admirable if they could divert their brain power in improving the radio system and presenting to the world not only the immediate talks, as we get at present, but speeches of past days, months, years or even ages and setting the radio to catch the instructions and preachings of past saints and godly men, like Jesus, Mahomet, Buddha and Srikrishna.

CASE NOTES

A CASE OF PNEUMOCOCCAL MENINGITIS WITH RECOVERY

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AND
C. M. VAILLANT, LIEUT-COL., R.A.M.C.
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Recovery in pneumococcal meningitis is exceptional and when the diagnosis is made one feels justified in adopting almost experimental forms of treatment.

It is clear that treatment should commence as early as possible and that the drugs used should be in adequate dosage and rapidly absorbed. The question arises, what is the adequate dosage?

Successful treatment implies not only the saving of life but the prevention of distressing sequelae. In one case reported to us verbally, Penicillin by itself (amount given not stated) was successful in preserving life but the brain was severely and permanently damaged after a slow recovery. Furthermore, for general use the technique must be simple. Penicillin injected into the subarachnoid space by cisternal or lumbar puncture has been reported on favourably in suppurative meningitis but it can be difficult or impossible to withdraw sufficient cerebrospinal fluid before introducing the penicillin.

Sulphapyridine in doses sufficient to cure meningococcal meningitis has never been, in the writers' experience, successful in pneumococcal meningitis and there is a tendency to regard it as too toxic to give in larger doses.

Our experience with sulphamethazine is limited to one case only in which it was unsuccessful. The intravenous preparation, the best form for use, was not, however, available.

Our justification for reporting the present case, in which intravenous sulphapyridine was used in combination with intramuscular penicillin, is that the result was immediately successful and a comparatively large amount of sulphapyridine given within a short time was tolerated without ill-effects.

CASE REPORT

An Indian labourer aged 22 and of good physique was admitted on 9-6-45 at 10.00 hours, having been unconscious for a few hours, with signs of meningitis. Duration of illness: 1 day. Pulse—66, respiration—16; temperature—100°F. Irritable, Brudzinski's and Kernig's signs present, exaggerated knee reflexes, flexor toes. Constipated. Other systems normal. Blood slide negative for malarial parasites. W.B.C.: 15,600. C.S.F.: Turbid and under slightly increased pressure. Cell count—14,000; chlorides—675, sugar—decreased. Pneumococci and pus cells present.

Treatment at 12.00 hours:

Simple enema—(1) Dagenan—2 gm. intravenously, followed by 1 gm. at intervals of 3 hours.

(2) Penicillin, 40,000 Oxford units, by intramuscular injection, to be given with (1).

Total day's dosage—Dagenan—6 gm., Penicillin —200,000 units.

10-6-45—General condition satisfactory. Temperature: 99·4°F.; respiration—16; pulse—62; blood slide negative for malarial parasites.

Total day's dosage—Dagenan—8 gm., Penicillin —320,000 units.

11-6-45—Patient conscious, able to walk. Complains of slight frontal headache. W.B.C.—12,000. Temperature normal and remained so thereafter. Later in the morning he performed a cartwheel to demonstrate his well-being. Urine—normal.

Treatment—(1) Dagenan stopped. (2) Penicillin continued. (3) Sulphapyridine: 1 gm. every 4 hours starting at 10.00 hrs. (By mouth).

Total day's dosage—Dagenan—2 gm., Penicillin —320,000 units., M. & B. 693—4 gm.

12-6-45—Appears normal. Pulse—76; respiration—18.

Treatment—(1) Penicillin stopped. (2) M. & B. 693 continued.

Total day's dosage—M. & B. 693—6 gm.

13-6-45—M. & B. 693—6 gm. given during the day.

14-6-45—All treatment except alkalinisation stopped.

Total amount of drugs given during the illness:

Dagenan—16 gm., Penicillin—840,000 units, M. & B.—16 gm.

18-6-45—Quite recovered, afebrile for 1 week. W.B.C.: 8,600.

2-7-45—Discharged to duty.

OBSERVATIONS

(1) The apparent effectiveness of increased dosages above the generally accepted amounts.

(2) The rapid response to treatment with absence of complications.

STANNOSULPHAZIDE AND CHAULMOGRA OIL IN CHRONIC SKIN ULCERATIONS

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Hereunder I am giving details of two cases of chronic ulcerations of the skin. The cases were treated with stannosulphazide with chaulmogra oil locally and stannosulphazide orally.

The results were not only encouraging but magical.

I must point out that though I am not a skin specialist and hence my experience should be taken only as of observatory nature specially as I did not go into the microscopical examination of the discharges and other investigations of which the poor patients could not bear the expenses. It depended solely on macroscopical appearances and a follow up of the case for some time.

CASE REPORTS

Case 1—Laj, aged 3½ years—Hindu girl—seen on 14-2-45—had ulcers covering the entire scalp for the last one year and a half. Ulcers small-sized with dried scabs covering them. Had been resisting all treatment. Scabs when removed revealed a pus-forming area with the shape of a crater.

Treatment—Head was shaved with a machine and the treatment started. Scalp washed with spirit soap (soft soap 1 dr., spt. methyl. 1 oz.). A cream composed of stannosulphazide 3 tablets, chaulmogra oil 2 dr., eucerine ad. 1 oz. was applied and the scalp covered with lint and cotton wool and bandaged.

In cases when it is not desirable to shave the head, I would suggest preparation of the following cream for convenience.

R/Stannosulphazide	..	3 tablets
Chaulmogra oil	..	2 dr.
Cocoanut oil	..	3 dr.
Eucerine ..	..	3 dr.
Sandal oil ..	..	few drops.

Rub into the scalp after drying the hair, after a bath, with soap and water.

Orally the following mixture was administered:—

R/Stannosulphazide	..	6 tablets
Nicotinic acid	..	3 „
Gum acacia ..	..	q.s.
Glucose ..	..	q.s.
Aqua chloroform ad.	..	8 oz.

Divide into 16 doses. 4 doses a day—one every third hour.

Along with the mixture, the cream and spirit soap were given for changing the dressings at home once daily.

Result—After 4 days, and to my surprise, I found the scalp in a perfectly healthy condition.

The mixture was repeated for another 4 days and the cream and soap used for 12 days in all. Till to-day she is alright.

Case 2—Roshan Bibi, aged 10 years—Mahomedan girl—seen on 15-2-45.

She had ulcerations of the left side of the face, affecting left lateral half eyebrow, eyelids, entire cheek, left alae nasi, upper lip, left half and laterally affecting upto the front part of the ear, maxillary angle and finishing off at the chin in the centre. Raw bloody surface with greenish white crusts hither and thither and especially all around the ulcerated surface. No itching, no pain but the part was tender. Duration 2 years. Resisted all treatment.

Treatment—The ulcerated area was swabbed daily with lukewarm 2 per cent sodi bi-carb lotion, crusts removed, part dried and the same cream as in Case 1 applied on a piece of lint covered with cotton wool and bandaged and the following mixture was given orally.

R/Nicotinic acid	..	24 tablets
Nicotinic Acid	..	8 „
Gum acacia ..	..	q.s.
Glucose ..	..	q.s.
Aqua chloroform	..	16 oz.

Divide into 32 doses. 4 doses a day—one every 3 hours.

Result—In 8 days she was totally cured with no relapse of symptoms till to-day.

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STERILIZATION OF SYRINGES

We draw the attention of our readers to a letter published under correspondence in this issue. This is a letter from a mofussil practitioner who was called to treat a case of mumps with complications. The patient was being given glucose intravenously twice daily and one injection of glucose produced severe reaction in the form of rigor, high temperature and feeble pulse. Our colleague injected coramine in the left arm and gas gangrene developed at the site of this injection about 30 hours later. He declares that the syringe was twice cleaned with rectified spirit before the injection of coramine. The letter ends by raising two questions.. The first one should be noted and the second one is better avoided. He asks "what could be the mode of sterilization in case of injections given during emergencies, as hundred per cent of private practitioners sterilize their syringes with spirit in such cases when there is no time for boiling". Regarding the popular method described to sterilize a syringe, we believe, the statement is almost correct. There are many reports from continental Europe, where gas gangrene developed after injection with a syringe which spirit failed to disinfect. So the above incident, though unfortunate, is not uncommon as is believed by the doctor. The question which arises in this connection is—can rectified spirit perfectly sterilize a syringe?

In the process of injection the sterility of three substances are concerned *viz.*, the injectable medicament, syringe and the skin. Instances are not rare where an infection has been transmitted due to a questionable sterility in any one of the above three. Rightly or wrongly every time when such disasters occur, the technique of sterilizing the syringe has been blamed. We all know that the common method of sterilizing the syringe is imperfect and although rare, accidents of the nature described above, may occur. A committee of the Medical Research Council made a comprehensive investigation of this problem and published a document entitled "The sterilization, use and care of syringes"[1]. A short and instructive resumé of the memorandum appeared in the current medical literature section[2] of the *Journal* in October, 1945. It seems that this summary has escaped the notice of some of our readers. As the subject is important to the profession, we are giving below the recommendations of the Committee regarding the question raised by our correspondent.

The most important rule in case of injections is to keep separate all syringes once used for aspiration of septic materials from anywhere in the body. Whatever method of sterilization is used later, these syringes should never be used again to inject sterile medicament. The usual practice should be to keep enough syringes and to use them separately for different jobs. This may be difficult to manage in these days but will be easy when enough supply of syringes are available at a reasonable price. The safest way of sterilizing a syringe is to autoclave it or to expose it to a temperature of 160°C in a dry oven. The Record syringe, so commonly used in our country, is quite unsuitable for this method because the cement at the glass metal junctions may melt at such a high temperature. So the Committee advises to use all-glass syringe preferably according to a British Standard specification [B.S. 1263(1945)]. The all-glass syringe has also some additional advantages. It can be easily cleaned and there are no crevices in which traces of previous content may lurk. The ideal policy is to have enough of these syringes and to sterilize them in glass tubes by dry heat. Boiling is the next best way to sterilize a syringe. Unfortunately this does not kill sporogenous organisms unless sodium bicarbonate is added to the water. Moreover traces of alkali in the syringe may alter the efficacy of the medicament to be injected. The danger of infection by sporogenous bacteria is uncommon but not rare. A dry syringe is always preferable to a wet one and for the reasons enumerated above, the Committee does not consider the above procedure to be an ideal one. The hot oil method has also been found to be ineffective.

The Committee also conducted experiments to decide the value of chemicals in sterilizing a syringe. It was found that hypochlorites, phenols, chlorophenols, quaternary ammonium salts and detergents were "ineffective as disinfectants for syringes" because they might alter the medicament to be injected. Regarding disinfection of syringe with alcohol, the Committee found that alcohol acted best in a concentration of 75 per cent by volume in water and in an undiluted state it was totally ineffective. Alcohol acts only on vegetative bacteria and like all chermical agents has little power of penetration and thus cannot disinfect the crevices and corners of a syringe. So it can be used to sterilize a two piece all-glass syringe taken apart, against vegetative bacteria but useless in case of a glass metal Record syringe. Archer[3] has shown that alcohol is a better disinfectant for skin than many other but when absolute sterilization for instruments is necessary, it is unreliable.

STORY OF BLACK HOLE IN CALCUTTA

The *British Medical Journal* in its issue of the 6th April, 1946, has written an annotation on the Black Hole to discuss the etiology of death of the people who died (?) therein.

The incidence of Black Hole has been conclusively proved as 'a gigantic hoax'.[1] There was a conference of historians on the 24th March, 1916, under the auspices of the Calcutta Historical Society, held in the hall of the Asiatic Society, in which the Hon'ble Mr. F. J. Monahan, Mr. C. H. Little and Sjt. Akshay Kumar Maitra proved historically that the story of the Black Hole was a fabrication deliberately interpolated into history.[2]

[1] Medical Research Council, War Memorandum, No. 15 H. M. Stationery Office, 1945, Price 4*d*.
[2] *J. Indian M. A.*, **15**:27, 1945.
[3] ARCHER, G. T. L.—*Brit. M. J.*, **2**:148, 1945.
[1] LITTLE, C. H.—Bengal Past and Present, Vol. 11, series 21, pp. 75 to 104.
[2] MAITRA, A. K.—Serajuddula, Appendix p. 415, 7th edition.

If anyone is really interested in the scientific aspect of such incidences we would refer him to the writing of Mr. Justice Beveridge of Calcutta High Court in the *Calcutta Review,* April 1892, about an incidence in Amritsar on the 1st of August 1857 after the Sepoy Mutiny.

282 (Indian sepoys) were kept confined in a room out of whom 237 were taken out and shot one after another. The rest *i.e.,* 45 were kept locked up in a small cell overnight. Next day the doors were opened and "Forty-five bodies—dead from fright, exhaustion, fatigue, heat and partial suffocation were dragged into light"[3]. They were all stone dead!

[3] The Crisis in the Punjab, p. 162.

We might also refer to a similar incidence of more recent memory—that of the Moplah Train Tragedy—where a large number of Moplah prisoners were packed into a railway wagon—to meet a similar nature of death.

We want to point out that the two incidents mentioned above, will give material for further investigation regarding death from suffocation, heat stroke, dehydration and heat exhaustion like the disaster of the Bethnal Green tube shelter in 1943 and fire at Coconut Grove, Boston, in 1942.

It may be recalled that the Holwell monument raised by Lord Curzon in 1902 to commemorate the so-called Black Hole was dismantled and removed by the Government of Bengal.

(*Continued from page 356 col. 1*)

Cases showing a higher SR at 99°F are marked SR+. Cases showing no appreciable difference (up to 10%) at any stage during an hour are marked SR O. Cases showing a higher SR at 44°F are marked SR—.

Of 483 cases examined in this way 279 (57·7%) showed SR+, 109 (22·7%) SR—, and 95 (19·7%) SR O; they are divisible into three groups.

Group I—This comprised respiratory (including uncomplicated pulmonary tuberculosis), circulatory, digestive, nervous, and skin diseases, gonorrhœa, arthritis, etc., and consisted of 252 cases, 198 showing SR + and 54 SR O. Six cases of ascites with SR + appear in this group: three of them were cancer of the peritoneum, one Pick's disease, and one chronic portal thrombosis. *Group II*—The SR varied in this group; details are given in a table. *Group III*—This group always gave SR—and consisted of: Atrophic cirrhosis of liver—19 cases, kala-azar—9, amœbic liver-abscess—6, pulmonary tuberculosis with intestinal tuberculosis—3, primary intestinal tuberculosis—4, cancer of the liver (one primary and two metastatic)—3, abacute icterus gravis—2 and arsenical hepatitis—1.

SR—appears to associated with damage to the parenchyma of the liver.

The authur concludes by saying that the simultaneous examination of SR in warm and cold temperatures seems to have a considerable diagnostic and progmostic value.

Damaged Intervertebral Disks: Early Diagnosis and Treatment

Crisp (*Lancet,* **2**:422, 1945) writes that the lumbar intervertebral disks suffer trauma far more often than is generally appreciated, but in the absence of crural pain or radiographic changes the resulting lesion is often mistaken for sacro-iliac strain or fibrositis.

The condition may easily be recognised by the extreme and persistent lumbar spasm and the "tight" lumbar spine. Early diagnosis and treatment by immobilisation should considerably reduce the incidence of sciatica.

Cases which escape diagnosis until after the onset of sciatic pain may be divided into two groups, depending on whether the annulus fibrosus is partially or completely ruptured. The former group may be recognised by the fixed lumbar lordosis, the latter by the fixed lumbar kyphosis. Both groups usually respond well to immobilisation in a spinal jacket.

Clinically these cases are distinguished by extreme and persistent spasm of the lumbar portion of the erector spinæ muscle and hamstring spasm of less severity. When the patient stands erect, little abnormal is noted beyond a slight increase in the lumbar curve, but on his bending forward to touch the toes a remarkable degree of lumbar spasm becomes obvious. This is sufficient to prevent any forward flexion in the joints of the lumbar spine, there being movement only at the hip-joints and the upper dorsal region. Some hyperextension and lateral flexion, however, are usually possible.

Besides these clinical signs there are characteristic symptoms. The patient will complain of continuous aching lumbar pain which is aggravated by movement and coughing and often radiates into the groin. There will be tenderness to deep pressure over the sacro-iliac joint and the interspinous ligament of the affected segment. The sacro-iliac tenderness often develops first; this may explain deagnostic errors. Scoliosis is unusual at this stage; but, as the case gets worse, hamstring spasm increases on the affected side, and on bending forward the body inclines to the side of lesion.

The spine is immobilised accordingly in a plaster-jacket. Two cardinal rules must be observed if sucess is to be achieved: first, in applying the jacket no attempt must be made to correct the deformity, for experience shows that this only leads to increased pain; secondly, the plaster must be worn long enough for the torn disk to become soundly healed. Immobilisation for 6-8 weeks appears to be sufficient for the partially ruptured disk diagnosed shortly after the injury and without sciatic symptoms. The plaster should be applied with the patient standing in the position of greatest comfort.

When the condition is only diagnosed after the onset of crural pain, a longer period of immobilisation obviously becomes necessary, for the damage and reaction will be more extensive. Usually the condition is recognised while the rupture is still incomplete and before protrusion has taken place, the lumbar spine being in fixed lordosis; but in view of the added complication the jacket must be worn for at least three months.

Surgical treatment may be reserved for cases where the protrusion has not been reduced after a reasonable period of immobilisation, say, up to three months; also for recurrent protrusions, and for patients who still complain of severe pain long after the lumbar spine has reverted to its normal curve. In this last class the herniated nucleus has probably become fragmented, and the detached portion may be found lying loose in the thecal canal.

CURRENT MEDICAL LITERATURE

Albuminuria in the Young

It is almost seventy years since von Leube (*Virchows Arch.*, **72**:145, 1875) noted that 4% of apparently healthy soldiers had albumin in their urine, and a multitude of observers have confirmed his finidings; yet symptomless albuminuria remains a teaser for doctors examining candidates for the Forces or life assurance. Intermittent or even persistent albuminuria can undoubtedly be consistent with good health and normal longevity. On the other hand, it is not rare in insurance practice to encounter a case where symptomless albuminuria is sooner or later proved to have been an indication of grave disease. It is probably impossible to separate the significant from the insignificant with absolute certainty, and this perhaps explains why life assurance statistics, such as those analysed by Dublin in the United States, show that a supposedly benign albuminuria carries a less favourable mortality than a normal urine.

That albumin in the urine is compatible with physical performance of a high order was illustrated by Collier's (*Trans. Med. Soc. Lond.*, **30**:75, 1906-7) observation that every member of the Oxford boat of 1906 passed large quantities after the race. During the 1914-18 war MacLean (*Spec. Rep. Ser. Med. Res. Coun.*, No. **43**, 1919) observed that 5·8% of 50,000 soldiers in training had albumin in their urine, whereas in only 1·1% was there definite evidence of renal damage. In another last-war series of over 2000 soldiers of medical units living in billets McLeod and Ameuille (*Lancet.*, **2**:468, 1916) reported albuminuria in 2·9%, casts in 0·86%, and red cells in 0·66%. Bashford's (*Lancet.*, **2**:1304, 1926) findings in Post Office workers are as striking as any. Thirty male employees, in whom albuminuria had been noted on their joining the Post Office, were re-examined twice—once after 7-14 years' service and again 17 years later. At the first re-examination all were in good health and albuminuria was still present in 6. The 24 men who attended for the second re-examination all had satisfactory health records and only one still had albuminuria. In a group of 1,000 boys of 14 years albuminuria was found in 5·8%, though there was no evidence of nephritis in any, and Theobald (*Lancet*, **2**:1380, 1932) reported albuminuria in 6·7% of 5000 Post Office girls. Lyall (*Brit. M. J.*, **2**:131, 1942) who found albumin on more than one occasion in the urine of 110 (0·55%) of 20,000 British recruits in the recent war, classified the cases as orthostatic albuminuria (28%), 'albuminuria without nephritis' (20%), subacute nephritis (13%), subchronic or chronic nephritis (28%), and infections of the urinary tract (11%). His criteria for the orthostatic type were: (1) The albuminuria is transient, and the urine passed during rest is free from albumin. (2) The urinary deposit shows no pus cells, red-blood cells, or renal casts. (3) The urea concentration of the urine passed within 2 hours after taking 15 g. of urea reaches over 3% and the blood-urea is normal. (4) There is no history of illness, particularly a recent illness which might result in nephritis. (5) The blood-pressure is normal, and there are no clinical evidences of nephritis or anatomical abnormalities of the urinary tract.

In his second group one or more of these criteria was unsatisfied, yet there was no definite evidence of a renal lesion. It is in this group that the prognosis is most difficult to assess. In the United States Wolman (*Am. J. Med. Sc.*, **210**:86, 1945) has analysed the urinary findings in 22,000 inductees, all of whom had previously been passed as fit by medical officers at their enrolment. Albumin was found in 420 of this selected group at their first examination as inductees, but it was remarkable that the 27 men who developed pyuria, and the 17 in whom major disorders of the urinary tract were detected during the 1-5 months they were under observation had all had normal urine. Eight cases of pre-existent nephritis were discovered at the first examination and no more were detected later. The examination of a random sample of urine, therefore, though it will almost always detect the presence of nephritis, will not pick out men liable to that disease, as MacLean noted with trench nephritis in the last war; and it may well miss other chronic urinary disturbances as well as intermittent albuminuria.

What steps then should be taken when albumin is discovered in the urine of an apparently healthy young man or woman? First, the medical history should be obtained, particularly as regards scarlet fever and frequent attacks of tonsillitis. Secondly, a thorough physical examination is essential, including estimation of the blood-pressure and examination of the abdomen to exclude polycystic kidneys, hydronephrosis, and the like. Thirdly, further specimens of urine should be examined, including at least one passed in the morning immediately on awakening. The urine should be centrifuged and the deposit examined microscopically. If albumin is absent in the early morning specimen and there are no other adverse findings the condition may be regarded as orthostatic and presumed to have no prognostic significance. If albumin is constantly present in all specimens particularly if it is in fair or large amount—it must be considered significant, and, for example, the person should not be passed for life assurance at normal rates of premium. Other concurrent unfavourable features, such as over-average blood-pressure or abnormal findings on microscopical examination, are cumulative evidence of a lesion in the urinary tract, and would call for rejection for life assurance. If red cells or pus cells are found it may be wise to carry out intravenous pyelography, which may reveal an unsuspected hydronephrosis, but cystoscopy or retrograde pyelography is rarely justifiable on such evidence alone. Renal-function tests are unlikely to be helpful in apparently benign albuminuria, and their routine employment in every case where albumin is found in the urine is a waste of time and money. *Lancet.* **2**:566, 1945.

Polyneuritis after Jungle Sores

Ward and Mason (*Brit. M. J.*, **2**:252, 1945) say that in a recent campaign in the Burmese jungle many of the British and Indian troops developed multiple indolent ulcers, commonly known as "jungle sores". Some of those who had suffered from jungle sores contracted a peripheral neuritis later. Of 21 patients 11 were admitted complaining of symptoms referable to their neuritis, 9 because of malarial relapse and 1 with jungle sores. All had suffered from jungle sores but, except for patient 19, the ulcers were healed at the time of admission. Sixteen patients reported blurring of vision as the first symptom of nervous involvement. The average duration of blurred vision was four weeks. All patients noted disturbance of sensation. In 4 cases it synchronized with blurring of vision, but in the remainder it occurred three weeks later. Sensory symptoms consisting of tingling, numbness and coldness of the extremities were often severe enough to make the men report sick. In the milder cases these paræsthesias

were the only complaints; however, others later complained of difficulty in walking and writing. The picture in its most severe from was completed by progressive weakness of the limbs, spreading to involve hips and shoulders. In addition anæsthesia of the extremities, astereognosis and ataxia were present. From a shuffling walk with feet apart the gait deteriorated until walking was impossible. Examination of the nervous system in these cases showed the characteristic signs of a polyneuritis. Eleven of the cases were mild. In 7 moderately severe cases the disability was sufficient to impede the patient's everyday life. Only 3 patients became bedridden. The polyneuritis was not influenced by the vitamin B complex. Moreover the nutrition of all patients was good. The author suggests that a toxin, elaborated by bacteria infecting the ulcers, was the responsible agent. He thinks it most likely that the polyneuritis followed a diphtheritic infection of the jungle sores.

Sprue in India

Keele and Bound (*Brit. M. J.*, **1**:77, 1946) give in the following lines the summary of their observations on a clinical study of 600 cases:—

The sprue syndrome has been modified by war conditions in India, bringing into prominence certain aetiological and clinical features. Such aetiological points are: (*a*) There is a marked seasonal incidence reaching its peak in the month of June. (*b*) The condition is prevalent in Assam, North Burma, Bengal, Bihar, and less so in Bombay Presidency. Whether military or geographical factors have predominated in this incidence cannot be stated. (*c*) There is a surprising lack of positive relationship with dysentery regarding the monthly incidence and the incidence of dysentery previous to sprue, which is not greater than in normals but one still retains the impression that in individual cases dysentery may unmask the latent sprue syndrome. (*d*) Suppressed or latent malaria exacerabates sprue. In such instances eradication of malarial infection rapidly improves the sprue syndrome. (*e*) A fair or a dark complexion bears no relation to the incidence of sprue, but fair men form a majority of severe cases. (*f*) Acute sprue has developed on a well-balanced diet under jungle warfare conditions. Palatability of diet is as urgent a physiological need as calories.

The clinical picture of acute sprue has been contrasted with that of the classical disease, not often seen in this series. The syndrome following dysentery and subacute hepatic necrosis is descirbed, and emphasis laid on the borderline cases with glossitis or steatorrhœa dissociated.

The complete pictures of vitamin-deficiency syndromes have not been met with. Signs of A-deficiency were equivocal. Nicotinic acid deficiency has been more frequent than riboflavin deficiency. No evidence was found of deficiency of vitamins B, C, D and K.

Faecal Fats—the methods of collecting stools at present standard are inadequate. Three-day collections on known fat diet with quantitative measure of stool fat per day, are essential for accuracy. The degree of steatorrhœa has little prognostic value.

Blood changes have rarely been severe. With the hæmoglobin less than 12 g.%, hyperchromic anæmia was more frequent.,

Prognosis as to life is good, but as to function it is poor. There is reason to believe that a large percentage will progress into the classical form unless liver therapy is continued.

From the above description it is clear that in sprue the whole almentary tract from lips to anus is affected—the accent of the process falling on different regions at different times. We are entirely ignorant of the nature of this process, though we know it to be modified by liver, nicotinic acid, and riboflavin. Nor are we well informed as to the nature of the dysfunctions in digestion and absorption in this condition.

The frequency of remission and relapse and the dissociation of signs and symptoms make assessment of diet or drug therapy very difficult. Many more controlled observations are needed, and until these are forthcoming theorizing on sprue is like trying to do a jigsaw puzzle without most of the pieces.

Restless Legs

"A common but practically unknown disease", described by Ekhom (*Acta. med. Scand. Suppl.*, 138, 1945) under the title of "restless legs," occurs in two forms, one paraesthetic and one painful—and Ekhom's monograph is based on 154 cases of the former (34 servere) and 15 of the latter. In the first form the presenting symptom is paræsthesia of a peculiarly disquieting type often described by the victim as "crawling." These sensations are usually felt between the knee and the ankle, although sometimes in the thigh, and are accompained by a feeling of weakness. They are so persistant and disagreeable that they compel the patient to keep moving his legs or walking about to gain relief. The symptoms appear only when the patient has been resting, particularly soon after he has gone to bed. In mild cases they pass off in a few minutes, but they may persist for hours, interfering with sleep. Spontaneous remissions are common. In the painful form the pain, which may or may not be accompanied by paraesthesia, has the same distribution in the legs and may be very protracted. In neither group can any clinical signs be detected. The commoner, paraesthetic form, usually responds to carbachol in doses of 2 mgm. four to six times daily by mouth. The intermittency of the symptoms differentiates the condition from polyneuritis or subacute combined degeneration of the cord, while the absence of involvement of the fingers excludes acroparæsthesia.

A similar condition has been described by Allison (*Canad. M. A. J.*, **48**:36, 1943) under the characteristically transatlantic title of "leg jitters." This consists of a curious unlocalised restlessness in one or both legs which is said to be distinctly unpleasant. It usually comes on when the legs get warm in bed, and is relieved by getting up and walking about. Immediate relief is obtained by chewing gr. 1/100 of nitroglycerin, and for this reason it is thought to be vascular in origin. Ekhom places the "restless legs" he describes in the category of those "chronic diseases with exclusively or mainly subjective symptoms which embitter but do not endanger the patient's life," but he disputes the inevitable suggestion that they are neurotic in origin. Many clinicians will welcome this refusal to call a condition a neurosis simply because no organic lesion can be demonstrated. *Lancet.*, **2**:470, 1945.

Pain in Pectoralis Minor

The established case of angina pectoris is easily recognised, but the differential diagnosis in the early stages is becoming increassingly complicated. The latest condition to be described as simulating angina pectoris is strain of the pectoralis minor muscle, which, in the opinion of an American observer Mendlowitz (*Am. Heart J.*, **30**:123, 1945)

ranks second to psychoneurosis and the anxiety state as a cause of precordial pain in men of military age. In such cases there may be no history of trauma, and the presenting symptom is pain, usually in the midclavicular region of the chest at the level of the 3rd, 4th and 5th ribs, sometimes rediating to the shoulder but never down the arm. The pain may be intermittent, and it may apear to be related to exertion but careful investigation shows that this is only so if the effort involves movement of the upper limb. The pain can be reproduced by making the patient push the upper arm forward against resistance when the elbow is at the side and behind the axis of the body. There is tenderness over the pectoralis minor, maximal at the site of the pain, and injection of procaine at this site abolishes the pain. Heat, massage, and local rest usually bring relief within a few days, but in two cases the attacks of pain recurred over a period of years. It is suggested that the pain in due to rupture of the pectoralis minor at its insertion into the chest wall. This is by no means the first time that attention has been drawn to the rupture of muscle fibres or fibrositis in the differential diagnosis of angina pectories, but it seems to be the first time that this particular lesion has been incriminated.

Two facts should always be remembered in examining suspected cases of angina pectoris: that a full and careful history is essential for correct diagnosis, and that the pain of angina is substernal. *Lancet.*

Effect of Large Doses of Alkali on Kidney Function

Army Malaria Research Unit, Oxford, (*Lancet,* **2**:701, 1945) gives in the following lines the summary of its observations on the effect of large doses of alkali on kidney function:

Three subjects were given an intensive course of alkali consisting of sodium bicarbonate gr. 60 and sodium citrate gr. 60 2-hourly. One subject received this course for 1 day; two subjects for 3 days.

All the subjects on alkali had disturbances of renal function. In one, on the course for 3 days, this was well marked and led to a three-fold increase in blood-urea.

All the subjects on alkali developed fairly intense alkalosis, not entirely compensated, together with sodium and water retention. There was dilution of the blood by about 10%.

Both the subjects on alkali for 3 days complained of symptoms.

A fourth subject was given an equivalent amount of sodium as sodium chloride for 24 hours. He showed a slight degree of sodium and water retention and no alteration of renal function.

The amount of alkali given in these experiments had measurable physiological effects, including disturbances of renal function.

We consider, therefore, that large doses of alkali should not be administered in conditions where renal failure may supervene.

Acid-fast Organisms in Gastric Resting Juice

Yates (*Brit. M. J.,* **2**:530, 1945) writes that in 4·6% of resting juice specimens examined, acid-fast organisms were present. These organisms were also alcohol-fast. As pointed out by Van Diense and Hooghiemster alcohol resistance of an acid-fast organism is not sufficient to identify it conclusively as the tubercle bacillus. In this case they proved to be saprophytes, ingested very possibly with fruits and vegetables, on which they are commonly found.

It appears, from the cases investigated, that under normal conditions the chance of tubercle bacilli being found in the gastric resting juice of non-tuberculous patients is very remote.

Since the examination of gastric washings for the tubercle bacillus is a valuable diagnostic procedure it should be noted that mere films where acid-fast bacilli are few and possibly deformed in appearance may perhaps be misleading without further investigation.

Treatment of Diabetic Acidosis and Diabetic Coma

Almy and Others (*J. A. M. A.,* **129**:863, 1945) observe that patients with diabetic acidosis or diabetic coma can be successfully treated with small standard and frequently repeated doses of insulin, that the administration of glucose in this disorder is beneficial and not harmful, that it is not necessary to follow the blood sugar and carbon dioxide combining power as guides to therapy, and that with such a simple method the constant attendance of specialists in diabetes mellitus is, in all but rare instances, unnecessary.

Interns are instructed to treat diabetic acidosis and diabetic coma as follows:

1. As soon as the diagnosis is made, 25 units of regular insulin are given subcutaneously and repeated every half hour until the patient is free from all signs of ketosis, as judged by clinical and laboratory evidence.

2. When possible, replacements of fluid, salt and carbohydrate should be given by mouth as follows: In each hour is given (*a*) 50 to 75 gm. of glucose as orange juice (500-750 c.c.), grape juice (300-450 c.c.), or fortified fruit juices; (*b*) salty broth, 200 c.c.; (*c*) enough water to make the total fluid intake 1,000-1,500 c.c.

3. If the patient is drowsy or comatose, or if the patient has vomited or has been nauseated during the preceding twenty-four hours, therapy must begin by the intravenous route: (*a*) Infusion of 5 per cent glucose in isotonic solution of sodium chloride, which should run in at first at the rate of 750-1,000 c.c. per hour. (*b*) The rate of this infusion should be reduced as soon as dehydration is relieved. Patients over 40 years of age, any with notable arteriosclerosis and any with cardiovascular disease may have to be infused at slower rates and should be observed frequently and with extreme care for signs of increased venous pressure and passive congestion of the lungs.

It is advisable to return to the oral route as soon as the patient is fully conscious and free from nausea.

4. Urine specimen is tested every half hour or every hour for sugar, acetone and diacetic acid, (by catheterization, if necessary). The foregoing therapy is continued until the urine is free from acetone and diacetic acid. The intern should not leave the ward until this is accomplished.

5. When ketonuria and all clinical signs of acidosis have disappeared, fruit juice and 25 units of insulin are given at two hour intervals for at least eight more hours.

6. If the patient is anuric, the therapy is guided by frequent estimations of the carbon dixide combining power of the blood. This rule will also apply if acidosis should occur without the presence of acetone bodies in the urine. (Such cases are exceedingly rare and have not yet occurred in our experience).

Untoward Reactions Attributable to Atabrine

The following general statements regarding atabrine and reactions attributable to atabrine are submitted for the information and guidance of all concerned:

1. The military value of atabrine in suppressing vivax malaria and curing falciparum malaria far out-weighs untoward effects which have been attributed with reason to the use of the drug.

2. Suppressive doses of atabrine greater than 0·7 gm. per week should not be employed routinely. This amount has been shown to provide adequate protection against clinical attacks of malaria provided "atabrine discipline" is strictly enforced. In clinical treatment of malarial attacks with atabrine, routine dosage should not exceed 2·8 gm. in seven days.

3. Atabrine suppressive medication should be discontinued promptly and atabrine should not be given for clinical treatment when persons develop a typical lichen planus, unexplained chronic eczematoid dermatoses, unexplained toxic erythematous eruptions, exfoliative dermatitis, severe leukopœnia, agranulocytosis and aplastic anæmia, acute hepatitis (not including disturbances believed to be due to malaria) or toxic psychoses which can be reasonably attributed to atabrine after careful clinical study.

4. It should be remembered that drugs other than atabrine, such as the sulphonamides and arsenicals, may be harmful to persons with the conditions mentioned.

5. Caution should be exercised in attributing disease conditions to atabrine until careful and complete studies have been made over a period of time to establish such relationship. Because of the widespread use of atabrine, its administration inevitably coincides with many diseases with which the drug has no connection. Even if a connection is established between atabrine and a given untoward effect, its significance relative to the military value of atabrine requires evaluation. It should be remembered that, since the use of atabrine became widespread, clinical attacks of falciparum malaria have been almost eliminated and deaths from malaria have been extremely rare. There is no question of the general superiority of atabrine over quinine, both for supression and for clinical treatment.—*J. A. M. A.*, **129**:1092, 1945.

Influence of Temperature on Sedimentation-rate

Weingarten (*Lancet*, **2**:526, 1945) writes that the general statement that SR are higher in warm temperatures is not correct.

Westergren's method was used with all technical precautions. The readings were taken every ten minutes. Blood was withdrawn with as little venous stasis as possible, the patient having been kept fasting. Two SR determinations were carried out simultaneously, one at 99°F, the other at 44°F. These temperatures are conveniently obtainable in every laboratory in an incubator and a refrigerator respectively. In a sufficiently large number of cases (the first 200 of the series) the blood was divided and kept for an hour at 99°F and 44°F, and after thorough shaking two tubes were filled with chilled and two with warm blood. One of each was then put into the cold and hot compartments. Except for a very slight deviation in the first minutes, previous chilling or warming did not affect the SR. Special care was taken to conserve steady conditions, and the doors of the chambers were opened for a few seconds only every ten minutes.

(Continued at foot of page 352)

NOTES & NEWS

I.M.A. RESEARCH FELLOWSHIP

Capt. P. B. Mukerji, B.SC., M.B., F.R.C.S. (EDIN.), D.M.R.E. (CANTAB), F.F.R. (LOND.), Hony. General Secretary, Indian Medical Association invites applications for one Research Fellowship under the Indian Medical Association Research Fund of the value of Rs. 150/- per month. The fellowship is open to men and women and will be tenable for a period of not more than two years. The applicants must be medical practitioners whose names occur on the Provincial Medical Registers.

The applicants must furnish the following informations in their applications along with certificates of physical fitness and character.

(*a*) Full name; (*b*) Age and Sex; (*c*) Permanent address; (*d*) Details of academic career, including particulars of previous research work done by the candidate; (*e*) Particulars of the proposed research scheme. (*f*) Details of financial supports from other sources which the applicant will be in receipt of during the period of fellowship.

The note on the proposed research under item (*e*) should give (I) a resume of research work done on the subject, indicating the present state of the knowledge on the subject (II) details of the proposed research, indicating (*i*) the methods to be employed, (*ii*) previous experience in the use of these methods and (*iii*) the experiments to be carried out.

Twenty five typed copies of the note on the proposed research should be sent.

Applications must be forwarded through the Principal or the Dean of a Medical College or the Director of a Laboratory or Institute where the applicant proposes to work and must be accompanied by a letter from the forwarding authority that he has critically examined the details of the proposed research and that he is willing to guide and direct as far as possible the investigation and provide laboratory facilities and the contingent expenses for the particular line of work will be provided by the College or the Institute concerned.

The progress of the work must be communicated every six months to the Convener of the Research Fund Committee, Indian Medical Association, 23 Hindusthan Buildings, Calcutta.

Typed applications should be addressed to the Convener, Research Fund Committee, Indian Medical Association, 23, Hindusthan Buildings, Calcutta.

COLLEGE OF PHYSICIANS & SURGEONS, BOMBAY

The College of Physicians and Surgeons of Bombay held F.C.P.S., D.G.O. and D.C.H. Examinations in April, 1946.

The following candidates have passed the Examination for the Diploma of Fellowship:—

Anilkant Chhaganlal Doshi, Kumudlal Chimanlal Gupta, Kundarpakant Tuljashanker Dholakia, Omar Valli Jooma, Winifred Catherine Braganza (Miss), Gool Nadirsha Modi (Miss), Eustace Joseph Sequeira, Kantilal Manmohandas Soparkar and Katayun Jivanji Udwadia (Miss).

The following candidates have passed the Examination for the Diploma in Gynæcology and Obstetrics:—

Pesi Behramsha Bharucha, Mahashweta Vithalray Bhatt (Miss), Purshottam Morarji Battani, Manilal Shobhagchand Shah and Taradevi Mukund Sonde (Miss).

The following candidates have passed the Examination for the Diploma in Child Health:—

Durga Govind Chitale (Miss), Usha Kanialal Munshi (Miss) and Jayantilal Mohanlal Shah.

THE INDIAN PHARMACIST PRIZE

A sum of rupees one hundred and fifty will be awarded to the author of a paper on "History of Pharmacy in India" considered the best, in the opinion of the *Indian Pharmacist*. The manuscript which in length should be about one hundred foolscap-size typed pages, is to be submitted to Prof. M. L. Schroff, Editor, The Indian Pharmacist, 7, Lower Rawdon Street, Calcutta, before the 15th November, 1946.

The *Indian Pharmacist* will retain exclusive rights to publish and own any or all of the manuscripts submitted.

BENGAL COUNCIL OF MEDICAL REGISTRATION

Excerpts from the proceedings of the meeting of Bengal Council of Medical Registration held on 12-2-46:

The qualifications of M.B., B.S., and L.M. & S. of Hyderabad were recommended to Government for recognition for registration on reciprocal basis of similar recognition by the Hyderabad Council of M.B., M.M.F. and L.M.F. of this province.

The following resolutions moved by Dr. A. D. Mukharji were carried:—

(*i*) In view of the acceptance of the unanimous recommendations of the Special Committee on Medical School education by the Government of Bengal and the decision to convert the Dacca and Campbell Medical Schools into colleges affiliated to the University this Council resolves that the other medical schools recognised by this Council be also raised to the college standard by the year 1950 at the latest, after which no school be allowed to admit students for the training of Medical Licentiates.

(*ii*) That the compartment system of maintaining the Register of the Medical Council, *viz.*, the Annual Medical List be replaced by publishing the list with titles of Registered practitioners irrespective of their qualifications as had been the practice before.

(*iii*) That the Government of Bengal be urged upon to undertake the amendments of the Bengal Medical Act as per various recommendations of this Council as they are more free now to take up normal work with the cessation of hostilities.

REFORM OF MEDICAL TRAINING IN THE U.S.S.R.

Medical schools in the Soviet Union have achieved considerable success, and the number of these schools and of the students attending them are expanding constantly. Pre-revolutionary Russia had but seven medical schools; now there are seventy-five. The number of students has now reached almost 100,000; and 23,000 new students were enrolled at the country's medical schools last autumn.

The successful work of these schools lies in the fields of both theoretical and applied medicine and during the war members of the staff were engaged on extensive clinical and research work. The government, aware of certain shortcomings still existing in our medical schools, has arrived at several decisions with the object of improving the training of our future physicians and surgeons, and with a view to incorporating the latest discoveries of medical science in the future doctor's training. Chief of these measures is to extend the requisite period of medical studies from five to six years.

The People's Commissariat of Health and the Committee for Higher Schools have approved the new curriculum drawn up in accordance with the six-year course. Here are a few examples of the changes introduced. The greater part of the study time in the first two years is occupied with anatomy, histology and embryology, biological chemistry, physiology and biology. The arrangement emphasizes the importance of these subjects as the bases on which theoretical medicine is built and developed. The 398 hours allotted to anatomy are distributed over four semesters, instead of over three, as in the former plan. Practical work in anatomy takes up twice as many hours as do lectures. The study of biology has been extended to cover three semesters, the first two being occupied with a detailed study of material in zoology and parasitology and the third with lectures on the basic laws of life (population, heredity, evolution, and so on).

Changes have been introduced into the study-plan of the third-year subjects, the most important being in the preliminary instruction in the treatment of internal diseases. In the new plan, the course is called 'Diagnosis, and Special Pathology and Therapy' The designation of the course points to its more concrete nature. The importance attached to this subject may be inferred from the increased number of hours allotted to it—332 hours in place of 220—and from the fact that the course now forms part of three semesters instead of two. Diagnosis is also studied in the fifth and sixth semesters, and specialized pathology and therapy in the seventh.

Special pathology and therapy are taught by means of lectures and demonstrations with patients. The course in diagnosis includes x-ray methods. Every chair of diagnosis and special pathology and therapy is to have ample x ray equipment, and the staff will include professors or lecturers specializing in x-ray analysis. In microbiology and pathological anatomy, the new scheme provides for increased attention to the practical side of these studies. Pathological physiology, however, is studied mainly in lectures, only a small part of the course being devoted to practical work.

Obstetrics and gynaecology are studied over four semesters in the new plan, the number of hours and their distribution aiming at a more complex and profound study of these important subjects. The student will acquire the theoretical knowledge required by the future practitioner, as well as practical experience. A new course, 'The History of Medicine'. has been included in the fourth year, with the object of improving the general educational level of the young doctor. The last three semesters are devoted chiefly to a study of hospital therapy, hospital surgery, children's diseases, contagious diseases and epidemiology.

Students who have successfully completed the preceding courses of study are permitted in their tenth and eleventh semesters to do independent work in hospitals, clinics and polyclinics, and are considered as interns in their twelfth semester, there will be elective courses in physiology, pathological physiology, pathological anatomy and clinical anatomy. The introduction of these courses will to a certain extent

make for the necessary connection between theoretical and practical medicine and the pathogenesis of disease.

The programme includes practice periods totalling four months, during which the students work in district and village hospitals and clinics.

The new programme includes a number of measures to improve the means of assessing a student's knowledge. There will be an increased number of subjects at the final state examination, including, for the first time, human anatomy. There can be no doubt that the new plan will make for better training and more highly qualified doctors in the Soviet Union. —*St. Thomas's Hosp. Gaz.*, **44**:58, 1946.

MEDICINE'S DEBT TO PASTEUR

Fifty years ago, on Sept. 28, 1895, Louis Pasteur died in his 73rd year. Though not a medical man he contributed more, both directly and indirectly, to the advance of medical science in the second half of the 19th century than perhaps any other man. The first half had seen the building up of solid knowledge of the correlation between physical signs and morbid anatomy, but of the causes of disease there was, as Osler said, "Before Pasteur, Egyptian darkness." In the retrospect one is struck with the beautifully logical development of his ideas, which grew from a minor point to wider problems. He was impressed by Laplace's vision of "a Galileo or a Newton arising through a series of inductions from particular phenomena to others more far-reaching," and this characterized all his work. Thus, trained as a chemist he turned to crystallography, and (incidentally creating stereo-chemistry) by his studies of dextrorotatory and lævorotatory tartaric acids, showed that only the former underwent fermentation, and that by means of living organisms—a thesis persistently denied by Liebig. When asked what was the use of his more recondite researches, he retorted by repeating Benjamin Franklin's question, "What is the use of a newborn child?"—a hint which might be taken by those present day scientists who would restrict scientific research to subjects of "social value."

Pasteur quickly showed the social value of his researches in crystallography by curing the diseases of beet sugar, wines, and beer; but what authority could have anticipated this? Moreover he said, "what would be most desirable would be to push these studies far enough to prepare the road for a serious research into the origin of various diseases (such as) putrid and contagious ones," believing as the far-sighted Robert Boyle had expressed it in the 17th century that "he who could discover the nature of ferments and fermentation would be more capable than anyone else of explaining the nature of certain diseases." Accordingly Pasteur started the study of silkworm disease, which was devastating the industry in these worms in France, and restored it to prosperity by discovery of the parasite responsible. He was bitterly opposed by the believers in spontaneous generation—a theory which it might have been supposed had been killed by Redi in the 17th century; but far from it, for this was the root of the opposition to the germ theory of disease. To do his opponents justice, they felt, as Piorry put it, "it is not the disease, an abstract thing, which we have to treat but the patient whom we must study with the utmost care." Their mistake was to deny the germ theory entirely; if they had not done so they might have realized that Pasteur himself had presented them with a proof that the soil mattered as well as the seed when he showed that animals naturally immune to an infection lost that immunity if placed under unfavourable conditions. Undeterred by opposition Pasteur went on to investigate the epidemics of "splenic fever" which were destroying valuable flocks and herds throughout France. He proved that they were due to infection by anthrax and perpetuated through the action of earthworms bringing to the surface the bacilli and spores from the buried bodies of animals that had died from the disease. His studies of chicken cholera led him to the important discovery of methods of attenuation of viruses and thus to the preparation of vaccines. "Is it not permissible," he said, "to believe by analogy that a day will come when easily applied preventive measures will arrest the ravages" of epidemic diseases? A dictum of his that "the best proof that an observer is on the right track lies in the uninterrupted fruitfulness of his work" was now to be strikingly illustrated by the application of his principle of attenuation to the treatment of hydrophobia. Early impressions persist, and one of Pasteur's childish recollections was the terror inspired in the Jura by the inroads of a mad wolf. He therefore attacked the problem with great energy and, as is well known, with great success, to which the foundation of the Pasteur Institute in Paris by public subscription was a striking witness. Here, with the assistance of Roux, Yersin, Chamberland, and Metchnikoff, his beneficient work continued and expanded, giving rise to similar institutes in various parts of the world. But there is no institute for hydrophobia in Great Britain, for the muzzling order stamped out the disease here. Pasteur's last years were rejoiced by the discovery of diphtheria antitoxin by Roux and Yersin at the Institute, and shortly after by Yersin's discovery of the plague bacillus in India. But perhaps his greatest pride in his followers was for Lister, whose earlier adaptation of Pasteurian methods to the control of sepsis after operation is recognized throughout the world as one of the greatest boons to suffering humanity—a striking instance of the fruitfulness of a single idea faithfully pursued.

Pasteur's work, here rapidly sketched, was the measure of the man, patient and indefatigable, remorseless in his criticism of his own findings, single-minded in pursuit of truth, and devoted to the welfare of mankind and therefore a firm believer in the internationalization of science. No man can have met with fiercer or more bitter opposition than this great benefactor. It is to be regretted that he spent so much time confuting adversaries who had only the flimsiest evidence to support them; but for one of his temperament it was impossible to leave the battlefield against ignorance just as it was impossible to deny pity and help to real suffering. A hard hitter in controversy, he had also a gift for loyal and devoted friendship. Perhaps his creed was best expressed in words from his oration at the opening of the Pasteur Institute—words which are even more relevant to-day than when they were spoken: "Two contrary laws seem to be wrestling with each other nowadays: the one of blood and death, ever imagining new means of destruction and forcing nations to be constantly ready for the battlefield; the other a law of peace, work, and health, ever evolving new means of delivering man from the scourges which beset him..... The latter places one human life above any victory, while the former would sacrifice hundreds and thousands of lives to the ambition of one..... Which of these two laws will ultimately prevail God alone knows."—*Brit. M. J.*, **2**:430, 1945.

CORRESPONDENCE

The Editor is not responsible for any views expressed by contributors

PROGRESSS OF SURGERY IN THE PROVINCE OF MADRAS

Sir,—We wish to draw your respectful attention to a reference made by Lt.-Col. T. S. Shastry, in an article "Progress of surgery in the Province of Madras", published in the November issue of the *Journal I.M.A.*, about our hospital. The information given is somewhat vague and incorrect. Instead of "Dr. Raju and his partner have an ambitious nursing home in Palakol, West Godavary" it may be read as "Drs. R. N. Raju and P. Ramarao are running a nursing home by name 'Osler Hospital', in the village of Podur, West Godavary District". We trust, that the minor change in the language will not infringe on the propriety of the author, but will remove the incorrectness. We have already communicated this matter to Lt.-Col. T. S. Shastry, but perhaps being busy with the Medical Mission to Malaya, he might not had time to communicate to you. I am etc.

Podur, West Godavary Dt. R. Narasimha Raju.
11-2-46.

EOSINOPHILIC CELLS

Sir,—Many 'stimulating' articles are appearing in the Journal on Eosinophilic leucocytosis. As a student I am writing a few lines about the same.

Eosinophil leucocytes develop as coarsely granular leucocytes from 'mesamœboid's of Minot or according to Ching Ching Wung from the ectoderm.

Eosinophiles may be regarded as on-lookers only in the body fight against the invading organisms. The increased percentage in the blood of eosinophiles is a sign of low vital resistance of the body. Their number is increased in various pathological conditions of the body *e.g.*, trichiniasis, many parasitic diseases, bronchial asthma, scarlet fever, many skin diseases and in certain pulmonary pathological conditions *e.g.* eosinophilic pseudo-leukæmia, lecithinophilic eosinophilia, eosinophilic lung and pseudo-tuberculosis with eosinophilia.

The treatment varies according to the cause.

Whether eosinophilic leucocytosis is an allergic manifestation or due to vagotonia is a debatable matter.

Estimation of serum fibrinogen, serum globulin and serum albumin is a helpful guide for the determination of allergy (increase of the first two and diminution of albumin). Examination of two blood films for differential count one before the injection of atropine sulphate gr. 1/100 and another within two hours from the time of injection is a valuable guide for the determination of vagotonia.

My view is that the eosinophilic leucocytosis is due to both allergy and vagotonia. My idea may be erroneous. I am etc.

Calcutta,
3-346. T. C. Bhattacharrya, M.B.

STERILIZATION OF SYRINGES

Sir,—I shall be extremely thankful if you kindly give me your valued opinion about the following case as it raises an important issue from the point of view of general practitioners all over India.

A patient aged 22 years suffering from mumps developed about the fifth day septicæmic type of temperature with orchitis, intractable vomiting, pain in the epigastrium and eyeballs, frequent rigors, with temperature ranging between 101° to 104°F.

He was being given intravenous injections of glucose 25 per cent 20 c.c. morning and evening injected in about 20 minutes' time.

The third injection caused severe reaction with rigor and shooting up of the temperature. Pulse became feeble and an injection of coramine was immediately given in the left arm. The patient developed gas gangrene at the site about 30 hours later.

The syringe and needle were sterilized twice with rectified spirit, once by the assistant and once by the doctor just before giving the injection.

In the circumstances:—

(1) Could the doctor be made liable; if so what else could be the mode of sterilization in case of injections given during emergencies, as hundred per cent of private practitioners sterilize their syringes with spirit in such cases when there is no time for boiling.

(2) Could gas gangrene be caused by the trauma of injections at the site by infection from the gall bladder secondary to mumps, being thrown out into the circulation by the severe reaction caused by the glucose injection.

I shall be very thankful if you kindly let me have your valued opinion regarding the above two points. I am, etc.

6-5-46
Latehor P.O. S. A. Mozaffer, M.B. (Cal.)
Dt. Palama, Bihar.

[*See* Editorial Comment—*Ed., J. Indian M. A.*]

WAS PENICILLIN (?) UNKNOWN IN INDIA?

Sir,—I beg to present the following lines and am inclined to believe that they deserve to be published in the Journal.

Several years before the Second World War broke out a Sanyasi remedy came to this region—a panacea for all ailments.

The thing was called a bread. This bread was to be put in a new earthen pot containing a concentrated solution of date *gur* (treacle obtained from the juice of date palm) for a number of days till it doubled. Of the two pieces of bread thus produced one was to be given over to some other patient and the liquid left in the earthen pot to be taken orally in specified doses for a few days.

A good number of patients suffering from diverse diseases were said to have been benefitted by the nostrum. From these facts it was realised that bread was undoubtedly some sort of fungus.

That a material elaborated in the process of growth of a fugus could have any healing properties was then not conceived. Moreover with our usual despicable apathy for such remedies no serious notice of the thing was taken.

For a year or so the remedy was much in vogue in the locality but afterwards disappeared completely. This disappearance seems quite natural in view of the fact that maintaining the continuation of subcultures was not an easy affair. The thing might have been some Penicillium or some other mould. It might still exist any where and if any medical man or a lay gentleman ever hears of the existence of any such substance within his reach, he should make earnest endeavour for procuring a specimen for proper scientific study of the mould.

16-5-46 Mohini Mohan Ray, M.B.
Kotalpur, Bankura

REVIEWS

SCOPE OF CHEMICAL INDUSTRY IN INDIA—by Hara Gopal Biswas, M.Sc. Published by the Bengal Chemical & Pharmaceutical Works Limited, Calcutta, 1945, Royal 8 vo. pp. 43. Price Re. 1/4/-.

In a pamphlet of 43 pages Mr. Biswas, (Chief Research Chemist, Bengal Chemical & Pharmaceutical Works Ltd.) has given his views on the 'Scope of Chemical Industry in India'. Coming as it does from an industrial chemist of some years' standing, the pamphlet is interesting and original in a number of directions. It is not of course expected that in a small pamphlet like this the subject of chemical industry can be sufficiently covered, but a perusal of Mr. Biswas's booklet would convince everybody that there is room for scientists and industrialists in this direction. India is proverbially rich in her potential resources and it is agreed on all hands that every effort should be made to exploit those resources. Many industrialists have not however a clear conception of the ways and means of getting into the chemical industry and Mr. Biswas's pamphlet, if read by them carefully, offer a stimulating exposition of the subject.

Mr. Biswas has divided chemical industry into two principal groups—inorganic and organic chemical industry. Under inorganic chemistry (heavy chemical industry) he has dealt with mineral acids, salts and alkalis, metals, etc. Under organic industry he has classified his text into (*a*) acids, alkaloids, sugars, essential oils of vegetable origin, (*b*) fermentation industry, (*c*) Coal tar distillation products and allied industries and (*d*) synthetic organic chemicals of aliphatic group and the industries dependent upon them. In addition to the usual items falling under these groups, the author has also dealt with a problem of laboratory glassware, artificial silk, styrene, plastics, synthetic rubber, and also hydrogenation of coal. The subjects dealt with, though in a nutshell, show the wide range of the author's interest and the tremendous possibilities that exist only for the intelligent industrialists to take up and harness the resources to the benefit of the country. We commend the pamphlet to the notice of those in this field of work and also to students of applied chemistry.

B. B. Roy.

MODERN ANÆSTHETIC PRACTICE—Edited by Humphrey Rolleston and Alan Moncrieff. Second Edition, 1946. Demy 8 vo. Eyre and Spothis Woode (Publishers) Ltd., London. 12*s.* 6*d.* net.

This handbook is the natural outcome of the tradition of the *Practitioner* of publishing their symposium articles in book form. This edition has been throughly revised. It consists of 12 chapters each devoted to one aspect of anæsthetic practice and contributed by an authority on anæsthesia. The most useful chapters are on basal narcosis, spinal anæsthesia. Anæsthesia and analgesia in midwifery are interesting. Even continuous caudal anæsthesia finds a place, where Dr. Elam advises that for the present this should be reserved for institutions only.

We recommend this book to general practitioners and specialists alike.

(*Continued at foot of col. 2*)

OBITUARY

Dr. B. M. DAS GUPTA

In the death in the early hours of the morning of 25th May, 1946, of Dr. B. M. Das Gupta, late Professor of Protozoology and Director, School of Tropical Medicine, Bengal has lost an outstanding medical research worker.

Dr. Das Gupta started life as a Licentiate from the Dibrugarh Medical School and was a Sub-Assistant Surgeon in the Brigade Laboratory at Bannu. His fine technique and careful microscopic work brought him to the notice of Col. Mackie and later of Col. Knowles. He worked for a long time at the Pasteur Institute at Shillong as a colleague of Col. Knowles and later when Col. Knowles came to the School of Tropical Medicine, Dr. Das Gupta joined him as Assistant Professor of Protozoology in 1921. From this time until the end of 1944, when he retired, he was associated with the School of Tropical Medicine.

Dr. Das Gupta's work in the domain of medical protozoology brought him international fame. He visited America and England under the auspices of the Rockefeller Foundation and was well received by all the foreign savants in his line of work. At the time of his retirement he was invited to a post at the London School of Tropical Medicine, but failing health prevented him from accepting that honour. Dr. Das Gupta was the author of a book on Protozoology and has contributed a large number of papers on various aspects of medical protozoology and malariology etc., in Indian and foreign journals.

Dr. Das Gupta was, at the time of his death, the President of the Jhargram branch of the Indian Medical Association.

He is survived by his wife and two sons with whom we sympathise in their bereavement.

(*Continued from previous column*)

PSYCHOLOGICAL MEDICINE by Desmond Curran and Eric Guttman, Second Edition. 1944. Demy 8 vo. pp. 196. Published by E & S Livingstone Ltd., 16 & 17 Teviot Place, Edinburgh, Price 10*s.* 6*d.*

For all higher medical examinations, psychological medicine is essential, though for undergraduates it still forms a minor subject. Necessity for an 'introduction' to bigger books was felt for that reason by the Post-Graduates. This little volume admirably fulfils that purpose. The main emphasis put on the unity of mental and physical state is important. All successful practitioners unconsciously do psychological practice, and this little introduction might give an insight into the present movement of psychosomatic medicine. We heartily recommend the book to students, general practitioners and specialists.

ILLUSTRATIONS OF REGIONAL ANATOMY by E. B. Jamieson, 6th Edition, 1946. D'F'Cap 8 vo. pp. 320. Published by E. & S. Livingstone Ltd., 16 & 17 Teviot Place. Edinburgh. Price 70*s.*

This, the best illustrations of regional anatomy, has now come out as a complete volume, which should be in all anatomy departments and libaries of medical colleges. Students will do well to have the book in separate parts which they can easily carry to the dissecting room. The illustrations are accurate, and well printed and convey an exact knowledge to the reader.